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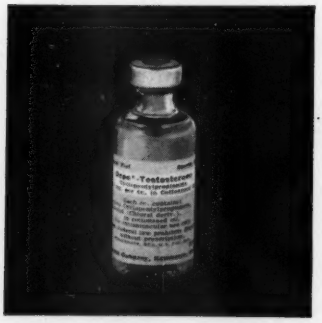

Entered at the Post Office in Baltimore, Maryland as second-class matter.

Louisiana State University School of Medicine Number

New Orleans, Louisiana

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THE
AMERICAN
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Vol. 20, No. 5 May, 1954

FOREWORD

The Louisiana State University School of Medicine, through its Department of Surgery, has been honored by the invitation to members of its staff to contribute to this issue of the American Surgeon. These papers, coming primarily from members of our Department of Surgery, are representative of the quality of the staff responsible for our teaching, research and service programs. It is important to note that the majority of the papers have been prepared by busy practicing surgeons, part-time staff members who have found time for a certain amount of investigation and who have in addition contributed greatly to our undergraduate, resident and graduate teaching programs. These part-time staff members bring to our students at the bedside and in the laboratories their experience in dealing with the practical problems involved in furnishing the best scientific and personal care for the individual patient.

The Louisiana State University School of Medicine is located in New Orleans, while the main campus of the university is in Baton Rouge, 80 miles from New Orleans. The School of Medicine which admitted its first students in 1931 was built adjacent to the Charity Hospital of Louisiana, one of the largest teaching hospitals in the United States. This large hospital offers all the clinical material needed to develop a sound program of medical education. The expansion of our teaching and research over the past two decades has caused the original physical plant to become inadequate. A new addition, almost equal to the original building in size, is now under construction. The original building has been remodeled, and our enlarged facilities will be ready for occupancy in August 1954.

The School of Medicine is a comparatively young division of the University. During the entire period of its development it has had the benefit of guidance by men of strong character, good judgment and integrity. One of the men who contributed most to the school's development is Dr. Urban Maes.* His ability as a medical educator, administrator, surgeon and scientist was responsible for

*Dr. Maes died on March 15, 1954, after this number of the American Surgeon went to press.

developing an outstanding Department of Surgery, and his contributions are recognized throughout the entire framework of the School of Medicine, in the local community and throughout the entire South. Many of the staff contributing to this issue are his students. When it became Dr. Maes' privilege to retire, Dr. James D. Rives was selected to head the department. Dr. Rives had served as a member of Dr. Maes' surgical staff for many years. Under the direction of Dr. Rives the Department of Surgery has continued to develop high standards of teaching and research in keeping with advances in surgery and the changing trends in medical education. Dr. P. Jorda Kahle, who was Professor and Head of the Department of Urology from the time the school opened until his retirement on June 30, 1953, makes his last contribution to medical literature in this issue. He had completed a case report just a few weeks before his death. Dr. Kahle was another of the staff who contributed much to the development of our School.

Although an adequate physical plant is a necessary part of our teaching and research activities, buildings do not set the standard of any educational institution. The educational standards are dependent upon the men and women who make up the teaching and research staff. The medical educator must have the ability and the initiative to adapt teaching methods and materials to the advances in medical knowledge and medical care. In surgery as in all aspects of medicine the best methods of today may be completely outmoded tomorrow. The vast body of scientific knowledge which is constantly accumulating must be gradually integrated into a comprehensive program of medical education. The selection of teaching material is a constant and ever recurring problem for the conscientious teacher. The surgeon, in his guidance of the medical student and the inexperienced resident physician, must teach technics and special skills without sacrificing basic scientific principles and scientific judgment. He must also teach the humanistic aspect of his subject, and when not to operate as well as when and how to operate. The new era of chemotherapy has made profound changes in the practice of surgery, as Dr. Rives has brought out in his editorial in this issue.

In addition to the teaching and research activities in the School of Medicine, our full-time and part-time faculty have responsibility for forty per cent of the medical in-patient and out-patient services of Charity Hospital. The average in-patient load is approximately 2700 and the average out-patient load is 2000 per day.

The School of Medicine is cooperating with other hospitals throughout the state and with medical societies in furnishing consultant and teaching services from our faculty. We are supervising a training program for interns and residents in one of our smaller State Charity Hospitals. Two surgical residents at Charity Hospital spend their entire fourth year in this smaller hospital, where they are supervised by our regular teaching staff.

The responsibilities of the Medical School faculty today are numerous and varied. Medicine is a learned profession which must be distinguished from other professions by the difference in its requirements. Good medical practice and

medical education must be interwoven with research. The valid new ideas and scientific developments must promptly find their way into our program of medical education. The well trained physician of today must be prepared not only to deal with a wide range of scientific evidence and technics but also to bring to his practice a broad understanding of human behavior.

The Louisiana State University School of Medicine is attempting to develop and maintain an educational and research program which will supply the best possible health and medical needs of our citizens. We are proud of the contribution which is being made to this endeavor by our Department of Surgery.

WILLIAM W. FRYE, Ph.D., M.D.
Dean
Louisiana State University
School of Medicine
New Orleans, Louisiana

MELENA

A SURVEY OF TWO HUNDRED AND SIX CASES*

JAMES D. RIVES, M.D., ROBERT O. EMMETT, M.D.

New Orleans

INTRODUCTION

In a previous study of 129 cases[2] of melena we expressed our concern about the fact that in one-third of these patients the cause for the bleeding was not found. We also stated that this figure was not a significant improvement over that reported by Harvey Stone[3] in 1944, in which the site of bleeding could not be located in 41.9 per cent of the patients.

The present report is a more comprehensive one, containing follow-up and other data on these 129 patients, together with 77 additional cases of melena. Included also in this consecutive series are two other categories of patients not previously considered.

MATERIAL

Over a 39 month period from March 25, 1950, to June 30, 1953, 5,026 patients were admitted to the surgical services of Charity Hospital at New Orleans for acute abdominal disease. Six hundred and thirteen (12.2 per cent) of these were hospitalized as emergencies for recent hemorrhage from the gastrointestinal tract. Of this group of 613 patients, 424 (69.2 per cent) had hematemesis with or without melenic stools. One hundred and eighty-nine (30.8 per cent) had melena alone. In addition, 10 patients developed melena during a hospital stay for some unrelated illness. Seven patients are also included in whom melena was disclosed only at autopsy. These three categories, totaling 206 patients, form the basis of this report.

The patients in whom melena occurred as a terminal event and was found only at autopsy represent a separate category. These 7 patients will not be included in the general statistics, but will be dealt with as an isolated group. There remain 199 cases for statistical study.

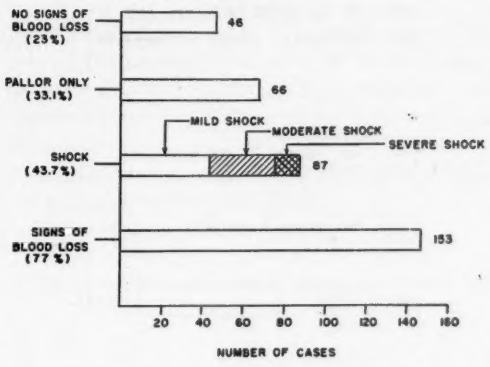
Any patient with hematemesis or with evidence of blood in the gastric contents was excluded from this series. Also not included are 15 cases of intussusception which occurred within the same period of time.

ESTIMATIONS OF BLOOD LOSS

Blood loss has been roughly estimated in these patients by routine red cell count, hematocrit determination, appraisal of the physical signs of blood loss, and the quantity of transfused blood required to correct the evidence of shock.

* From the Department of Surgery, Louisiana State University School of Medicine, and the L. S. U. Division of Surgery, Charity Hospital of Louisiana at New Orleans.

CHART I



As yet we do not have measurements of blood volume available as a routine procedure in this hospital.

Chart I reveals that 77 per cent of the patients in this study had physical signs of blood loss, and that 43.7 per cent were in some phase of shock on admission. In approximately one-half of the entire group of 199 patients the initial hematocrit reading was 30 or less. One hundred and twenty patients (60.3 per cent) required 2,000 cc. or more of blood to correct the signs of shock.

A constant observation during this study was the fact that the number of

CHART II

AGE INCIDENCE AND MORTALITY RATE IN 199 CONSECUTIVE CASES OF MELENA

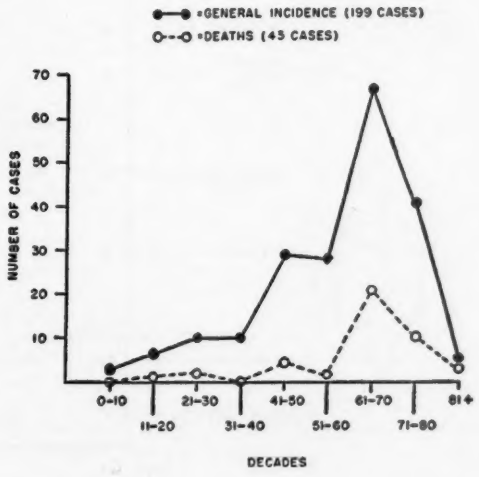
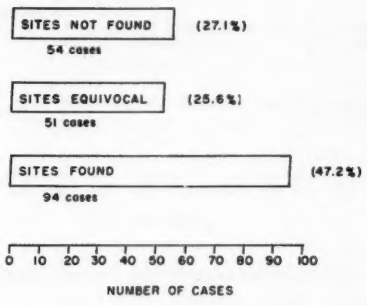


CHART III

SITES OF BLEEDING ENCOUNTERED IN 199 CONSECUTIVE CASES OF MELENA

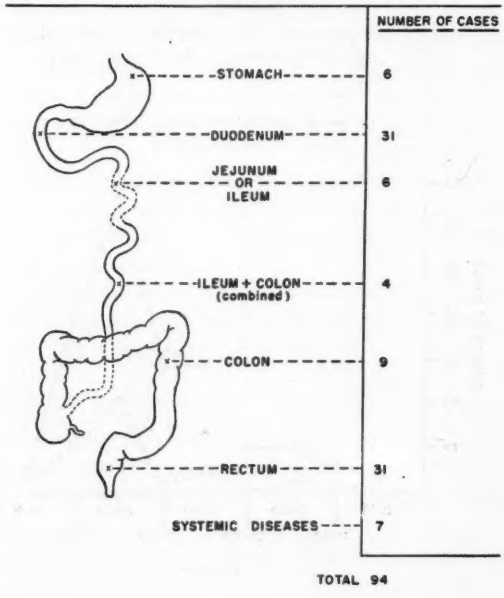


tarry stools passed per rectum did not afford even a clumsy estimate of the quantity of blood lost.

No relationship could be established between the severity of the hemorrhage and any particular type of lesion.

CHART IV

94 PROVEN SITES OF BLEEDING ENCOUNTERED IN 199 CASES OF MELENA



AGE, SEX, AND RACIAL INCIDENCE

The peak age incidence of melena in this series was surprising (chart II). Eighty-one per cent of the patients were over 40 years of age, and 55 per cent over 60 years. The occurrence of hemorrhage in those decades of life during which degenerative diseases are prevalent had a decided influence on the final outcome of the bleeding episode.

The preponderance of males in this series (62.1 per cent) could not be explained on the sex ratio of general hospital admissions.

No particular racial incidence was evident.

CHART V. 94 Proven sites of bleeding found in 199 cases of melena

	No. Cases	
Stomach		6
Carcinoma	4	
Ulcer	1	
Foreign body	1	
Duodenum		31
Ulcer	31	
Jejunum and ileum		6
Mesenteric thrombosis	2	
Tuberculous enteritis	2	
Nonspecific enteritis	1	
Meckel's diverticulum	1	
Ileum and colon		4
Nonspecific ileocolitis	2	
Tuberculous enterocolitis	1	
Mesenteric thrombosis	1	
Colon		9
Diverticulosis	5	
Carcinoma	3	
Amebic colitis	1	
Rectum		31
Carcinoma	6	
Hemorrhoids	5	
Acute proctitis	4	
Posthemorrhoidectomy bleeding	3	
Carcinoma of prostate invading rectum	3	
Postbiopsy bleeding	2	
Polyps	2	
Irradiation proctitis	2	
Rectal tear	1	
Simple ulcer	1	
Rectal stricture	1	
Carcinoma of uterus invading rectum	1	
Systemic diseases		7
Uremia	3	
Leukemia	2	
Anticoagulant bleeding	1	
Nonspecific hemorrhagic enterocolitis	1	

PROVED SITES OF BLEEDING

Reference to chart III shows that the source of bleeding was found in slightly less than one-half of the 199 cases of melena. We are deeply concerned about the other one-half in which the cause for the hemorrhage was either equivocal or not found.

It is noteworthy that a majority of the 94 proved sites were located in the most accessible portions of the gastrointestinal tract, i.e., the duodenum and the rectum (chart IV). Sixty-two lesions were demonstrated beyond doubt in these two locations. Of these 62 sites, duodenal ulcer accounted for one-half, and diseases in and about the rectum for the remaining one-half (chart V). It is particularly impressive to note that a wide variety of lesions, especially in the lower segments of the bowel, may produce alarming hemorrhage.

EQUIVOCAL SITES OF BLEEDING

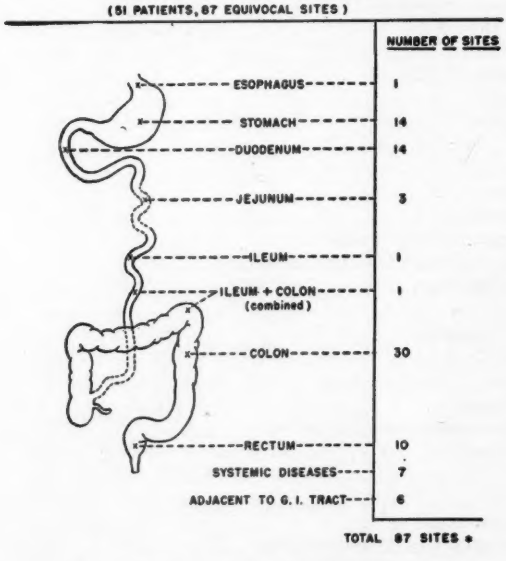
Chart VI lists the locations of the equivocal sites, and chart VII the specific lesions encountered in this category. Fifty-one patients had 87 possible sources of their melena. The preponderance of these equivocal lesions was in the gastroduodenal and rectosigmoid segments.

Worthy of special comment is diverticulosis of the colon, which was present in 28 patients in the equivocal group. The equivocation in these 28 patients was

CHART VI

EQUIVOCAL SITES OF BLEEDING ENCOUNTERED IN 199 CONSECUTIVE CASES OF MELENA

(51 PATIENTS, 87 EQUIVOCAL SITES)



* 34 PATIENTS HAD MORE THAN ONE POSSIBLE SITE

CHART VII. Equivocal sites of bleeding encountered in 199 consecutive cases of melena (51 patients—87 equivocal sites).

	No. Sites
Esophagus	1
Varices	1
Stomach	14
Hiatal hernia	5
Ulcer	2
Carcinoma	2
Gastritis	2
Prolapse of gastric mucosa	2
Diverticulum	2
Duodenum	14
Ulcer	8
Diverticulum	5
Duodenitis	1
Jejunum	3
Marginal ulcer	2
Diverticulosis	1
Ileum	1
Meckel's diverticulum	1
Colon	30
Diverticulosis	28
Sigmoid polyp	1
Nonspecific colitis	1
Rectum	10
Hemorrhoids	4
Polyps	2
Fissure	1
Simple ulcer	1
Acute proctitis	1
Fecal impaction	1
Diseases adjacent to gastrointestinal tract	6
Carcinoma of pancreas	1
Chronic pancreatitis	1
Cystic disease of pancreas	1
Cirrhosis of liver	1
Gallstones	1
Carcinoma of prostate invading rectum	1
Systemic diseases	7
Uremia	5
Unclassified bleeding tendency	1

not whether diverticula were present in their colons, but whether the bleeding actually originated from the diverticula. Over one-half of the patients with colon diverticula also had other possible bleeding lesions such as severe hemorrhoids, rectosigmoid polyps, and in one instance, a Meckel's diverticulum. Despite these multiple lesions we are reasonably certain that the colon diverticula were responsible for the hemorrhage in most, if not all, of these patients.

On the basis of a recent series of cases by Welch[4], the general incidence of bleeding in colon diverticulitis would appear to be about 27 per cent. Of a group of 76

patients admitted to Charity Hospital at New Orleans for complications of diverticulosis, Ochsner[1] found bleeding to be the most common reason for admission, occurring in 48.7 per cent of this group. These figures lend support to the progressive tendency of accepting diverticulitis as a common source of lower intestinal bleeding.

Notably uncommon in the entire group of 199 patients were lesions of the jejunum or ileum producing melena. Only 10 lesions were found in the small bowel which may have accounted for the bleeding. Four of these 10 sites were of an equivocal nature. Whether the scarcity of lesions in the jejunum and ileum represents an immunity of this bowel segment to bleeding lesions, or whether it reflects an immunity to satisfactory clinical examination, we do not know.

Another unexpected finding was the infrequency of polyps of the bowel as a cause of massive bleeding. Polyps were found in only 5 patients of the total number. In 3 of these whether the polyps actually caused the bleeding was doubtful. This is not to say that bleeding is not a common manifestation of polyps, but that in our experience it is seldom massive.

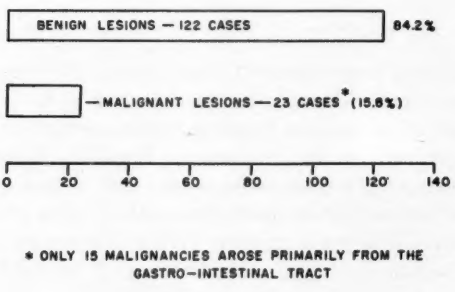
INCIDENCE OF MALIGNANT LESIONS

Of a total of 145 patients in whom the site of bleeding was definitely found or believed to be found, 23 patients had malignant lesions (chart VIII). In only 15 of these 23 instances did the malignancy arise primarily from the gastrointestinal tract. The remaining 8 patients represented carcinomas arising adjacent to the bowel or were instances of malignant blood dyscrasias.

Inasmuch as 85 per cent of the demonstrable lesions in these 145 patients were benign, we can state that (in this series at least) carcinoma of the gastrointestinal tract is not a common cause of massive melena. It was noticeable, moreover, that

CHART VIII

INCIDENCE OF BENIGN AND MALIGNANT LESIONS
IN 145 CASES OF MELENA
(PROVEN + EQUIVOCAL SITES)



* ONLY 15 MALIGNANCIES AROSE PRIMARILY FROM THE GASTRO-INTESTINAL TRACT

when a malignant lesion did produce severe bleeding it was usually a far-advanced lesion which was readily apparent to the examiner. We do not wish to convey the idea that bleeding is not of common occurrence in malignant disease of the bowel, but that it is usually not massive.

MORTALITY AND INCIDENCE OF ASSOCIATED DISEASE

The intolerance of elderly patients to acute blood loss was clearly demonstrated in the mortality figures in chart II. Ninety-three per cent of the total number of deaths were in patients over 40 years of age, whereas this group represented only 81 per cent of the total number of patients. Eighty-three and seven-tenths per cent of the deaths occurred in patients over 60 years of age, whereas this group accounted for only 55 per cent of the total number of cases. A total of 43 deaths gave an over-all mortality rate of 21.6 per cent.

The incidence of associated or degenerative diseases in the older decades of life was strikingly high in this series. One hundred and thirty-eight of 199 patients had 198 associated diseases of clinical importance, most of which were of a degenerative nature involving the heart and/or blood vessels. The exact relationship of hypertension, arteriosclerosis, and cardiac failure to gastrointestinal hemorrhage cannot be assessed with any accuracy, but few of us would challenge such predisposing factors as brittle blood vessels, passive liver congestion with mild secondary rises in portal tension, and the habits of constipation and catharsis in these elderly patients.

UNDISCOVERED SITES OF BLEEDING

In 54 patients (27.1 per cent) of 199, no explanation for the hemorrhage could be found. We have paid particular attention to this group in an effort to determine why the sites of bleeding could not be located. In spite of this, no useful observations can be made. This group of 54 patients, as compared to the other two groups, presented no appreciable differences in age, sex, or racial incidences. Nor was the severity of the hemorrhage or incidence of associated disease any greater.

UNSUSPECTED CASES OF MELENA

Seven cases occurred during the period of this survey in which the finding of melena was made only at the autopsy table. In this group of patients the gastrointestinal hemorrhage was so exsanguinating as to produce immediate death, or the melena was a terminal event in an otherwise irreversible illness. In our experience hemorrhage of the terminal type occurred most commonly in patients with severe jaundice, uremia, blood dyscrasias, and advanced carcinoma. In 1 instance an extensive carcinoma caused a fistula between the bowel and the vena cava. In 3 patients in this group acute gastric ulcerations occurred late in the course of a prolonged illness. In 2 of these, the use of a Levin tube for feeding purposes over a long period of time aroused our suspicions. In all patients in this category the prognosis before the onset of the melena was hopeless, and the terminal hemorrhage was more of a blessing to these debilitated patients than it was a problem to us.

FOLLOW-UP STUDY

Our follow-up study was concentrated on 105 patients in whom the site of bleeding was equivocal or not found. Seventy-six of these patients returned for outpatient visits, the remainder being lost for follow-up study. The average follow-up period for the 76 patients was nine months. Forty-five of those followed averaged 13.5 months. Of this entire group, only 5 patients experienced further episodes of melena, none of which could be classified as being massive.

Despite adequate and often repeated roentgenograms, laboratory, and endoscopic studies in the majority of these patients, only two sites of bleeding were discovered which had not been previously demonstrated. In addition, one equivocal site was found.

We are keenly disappointed in the results of this follow-up study in that only 2 patients of 76 benefited from outpatient visits and the repeated diagnostic procedures. We must acknowledge that our diagnostic tools are inadequate for certain segments of the gastrointestinal tract. Lesions in the gastroduodenal and the rectosigmoidal areas can be demonstrated with a high degree of assurance. Between these two segments, and particularly at the points of bowel flexures and fixations, are where improvements in diagnosis should be made. Endoscopic study of these inaccessible regions would be most desirable and accurate, but this is not a practical approach. More practical, but less accurate, would be the more frequent use of air-contrast barium studies in the small bowel, reaching it by using a long tube.

METHODS OF DIAGNOSIS

From the foregoing paragraphs it is evident that the problem of melena is essentially one of diagnosis. Definitive treatment usually is not difficult if the location and nature of the bleeding lesion is known. For this reason we have attempted to evaluate and tabulate the various methods which have been useful in our hands in arriving at a diagnosis.

History and Physical Examination. It has been our experience that a reliable history and a satisfactory physical examination have far surpassed any other method or methods of arriving at an explanation for melena. In 66 (70.2 per cent) of the 94 patients with proved sites of bleeding, the admission diagnosis was ultimately found to be correct. The majority of these tentative diagnoses, based on the examining senses alone, were made by admitting physicians or by surgical residents in their first or second year of training.

Previous History. A history of a peptic ulcer syndrome, or of a previous surgical closure of a perforated peptic ulcer, was found to be highly reliable in this study. In the 46 patients who gave such a history, there was only one instance in which this was misleading.

Quite the reverse was noted in the 41 patients who related a past history suggestive of a malignant lesion. Only a handful of the patients in this group were ultimately proved to have carcinomas. The explanation for this discrepancy probably rests with the frequency in which those diseases peculiar to geriatrics mimic malignant lesions.

Sixty-seven patients in the entire group related a history of having had previous episodes of melena. In those patients whose episodes occurred at wide spaced intervals, this information was helpful in that it suggested that a benign lesion was responsible. But except for this, the history of repeated bouts of bleeding was not found to be particularly useful in establishing a diagnosis.

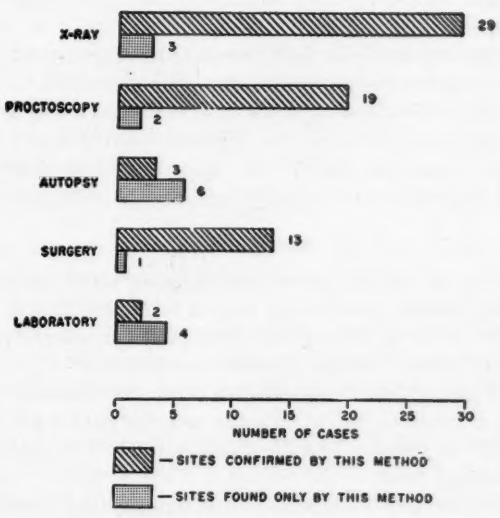
Color of Stool. Generally speaking, the color of the stool was of value in estimating the level of the lesion in most patients in this series. The majority of the lesions in the gastroduodenal segment produced tarry stools, and the majority of those in the colon and rectum caused maroon or red stools. Notable exceptions did occur, however, such as the 8 of 31 proved cases of duodenal ulcer in which the stools were maroon or red in color. The obvious conclusion is that, whereas the color of the stool is helpful in locating a bleeding site, it cannot be given too much reliance.

DIAGNOSTIC PROCEDURES

Chart IX roughly outlines the relative values of certain diagnostic methods by which the site of bleeding was determined in the 94 patients with unequivocal lesions. In only 16 instances did roentgenographic studies, the laboratory, proctoscopy, surgery, or autopsy discover a site of bleeding which was not suspected on a clinical basis. In 66 patients these procedures were of distinct value in confirming the clinical impression. These facts should serve to emphasize the

CHART IX

RELATIVE VALUES OF METHODS OF DIAGNOSIS IN 94 CASES OF MELENA (SITES PROVEN)



importance of an unhurried inquiry into the history of the present illness, and the value of a reasonably thorough physical examination. They are not meant to detract from the value of roentgenograms, laboratory and endoscopic studies. Such procedures are invaluable in accurately locating a lesion and routinely should be used as an essential part in the study of any patient with melena.

Surgery as a Means of Diagnosis. Surgical exploration has proved—in our hands at least—to be a very unsatisfactory method of finding a cause for melena. A total of 63 patients was submitted to surgery and in only 1 instance was a site of bleeding discovered which was not previously suspected on a clinical basis. This particular patient was one with a Meckel's diverticulum. In 9 exploratory laparotomies which were done in the hopes of establishing a diagnosis, no site was found. In 3 additional patients, an equivocal lesion was disclosed at surgery. In the remaining 50 patients who were operated upon, the diagnosis was evident before surgery.

DISCUSSION

In a previous communication we suggested that those lesions which could not be found probably were located somewhere between the gastroduodenal segment and the left colon. Subsequent observation and speculation has led us to modify this opinion.

Of the 13 patients who came to autopsy without an established diagnosis—7 of whom were not suspected of having melena clinically—the bleeding site was found to be in the gastroduodenal segment in 5 patients, generalized mucosal hemorrhage in 5 patients, in the small bowel in 2 patients, and in the colon in 1 instance. In light of these autopsy findings, we lean now to the speculation that in many of the unexplained hemorrhages the source may have been acute gastric ulcers which healed so quickly and completely that they could not be demonstrated by the roentgenogram.

We are encouraged in this belief by other isolated observations in which bleeding occurred from acute gastric ulcerations after prolonged vomiting or after prolonged use of indwelling gastric tubes. It is well known that acute ulcerations of the stomach are prone to occur as a terminal event in many debilitating illnesses, as well as during episodes of acute gastritis. These ulcers are fleeting in nature and may heal with extraordinary rapidity and completeness.

DIAGNOSTIC ROUTINE

From our experience with melena we have adopted a flexible method by which these patients are handled. Briefly, our plan of management is as follows:

1. An estimation of the severity of the hemorrhage is made and proper measures are taken to correct the evidences of blood loss and shock.

2. A finger is inserted into the rectum to verify the presence of blood, to ascertain the color of the stool, and to note any palpable pathology which may be of help. A Levin tube is inserted into the stomach to see if blood is present in the upper gastrointestinal tract.

3. A history is taken and a reasonably thorough physical examination is made.

In our experience this has far surpassed any other method or methods in establishing a likely cause for the melena.

4. Routine laboratory procedures are made, including a complete peripheral blood picture to rule out an unexpected leukemia or thrombocytopenia. Bleeding and clotting time and a tourniquet test for capillary fragility are done at the bedside.

Rush blood chemistries are made, including a determination of non-protein nitrogen or of urea nitrogen. An occasional case of unsuspected uremia may be found by this procedure. It is not likely that the rise in blood nitrogen due to the presence of large quantities of blood in the bowel will be mistaken for the higher rises seen in the uremic states.

5. If the evidence at hand suggests that the lesion is in the gastroduodenal tract, roentgenographic studies of this segment are made even though active bleeding is present. The presence of large clots of blood in the stomach and duodenum do not interfere with the radiologic technic as much as we had previously thought. We have come to believe that barium studies during the bleeding phase are of real value.

6. If the bleeding is thought to be coming from the lower bowel, a sigmoidoscopic examination should be done. Arrangements for a suction apparatus should be made beforehand in order to cope with the large quantity of blood and feces. In some patients, in order to remove clumps of feces and clots of blood, we have employed an ordinary rectal tube of generous size attached to the suction and inserted through the proctoscope.

7. If the information gained from the proctoscope reveals the blood to be coming from a point higher than can be reached by this instrument, then a barium enema should be considered. In the absence of peritoneal signs suggestive of a diverticulitis, and in the hands of a competent and cautious radiologist, such a procedure is safe and may yield valuable information.

8. Finally, if the hemorrhage is unrelenting and blood loss continues to exceed that which can be replaced with safety, one may be forced to resort to surgery as a diagnostic and life-saving procedure. We would again like to state that in our hands exploratory laparotomy as a means of locating a lesion which cannot be found by other methods is quite unreliable.

CONCLUSIONS

In a series of 199 cases of melena a cause for the bleeding was found in slightly less than one-half of the patients. In the other one-half, the cause was either not found or of an equivocal nature.

In those patients in whom the cause of the bleeding was definitely found or believed to be found, the common lesions were peptic ulcer in 48 patients, colon diverticulosis in 33 patients, and miscellaneous diseases of the rectum in 41 patients.

In 54 patients no cause for the melena could be demonstrated.

A satisfactory history and physical examination proved to be the most reliable method of establishing a likely cause for the hemorrhage in this series.

Roentgenograms, laboratory, and endoscopic studies were invaluable in confirming the clinical impression in many cases, and in detecting an unsuspected site of bleeding in some patients.

Surgical exploration as a method of establishing a diagnosis was found to be highly unreliable.

The infrequency of polyps of the bowel as a cause of massive melena and the scarcity of bleeding lesions in the jejunum and ileum were two unexpected findings in this study.

Eighty-five per cent of the demonstrable lesions producing massive hemorrhage in this series were benign. This figure would rise to about 90 per cent if we include those patients who, following an episode of unexplained melena, remained in good health for a period of time sufficient to rule out a malignant lesion.

The speculation has been made that acute gastric ulcerations, transitory in nature and rapid in healing, may be responsible for some, if not most, of the unexplained gastrointestinal hemorrhages.

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ACUTE INFLAMMATIONS OF THE CECUM

A STUDY OF TWENTY-THREE CASES*

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INTRODUCTION

Recorded in the literature are approximately 144 cases of solitary diverticulitis of the cecum[1, 5, 6, 9, 17, 19], 87 cases of simple ulcer[11], and 44 instances of primary *phlegmonous typhlitis*.[4, 12, 15, 16, 18, 21] These figures are misleading in that they suggest that inflammatory disorders of the cecum are clinical curiosities rather than practical problems. Inasmuch as these inflammations disguise themselves as acute appendicitis and are seldom diagnosed or even suspected prior to surgery, they constitute a group of lesions the recognition and management of which should be familiar to anyone who does appendectomies.

TERMINOLOGY

The most ill-defined of these lesions is acute *phlegmon* of the cecum. The current misconceptions about this disease are supported by a variety of synonyms which still appear in modern medical literature. Many of these synonyms, such as stercoraceous typhlitis, primary perforating typhlitis, parietal gangrene of the cecum, and cecum lignum, are based on the gross pathology as seen in isolated cases. Many false disease entities of the cecum have arisen and persisted as a results of these variations in terminology.

It is remarkable that the words *typhlitis* and *phlegmon* have survived the changing trends of several generations of medicine. Both bear the scars and stigmas of many medical controversies, and it is generally agreed that one is lacking in having any distinct pathologic definition and that the other is shrouded by historic implications. If one accepts these terms as being obsolete or on unsteady pathologic grounds, then their continued use in medical literature is hardly justified other than for historic purposes.

The term *cecitis* will be used throughout this report in an effort to standardize the terminology. The cases to be discussed are those with acute inflammation in the wall of the cecum with or without abscess formation.

It is imperative to state that the lesions to be presented are not granulomas or of granulomatous character, but are acute in nature and recent in origin. Of chief concern, therefore, will be those inflammations which spread to the cecum by *continuity* from the walls of those organs which are continuous with the cecum, or those inflammations which are thought to arise *primarily* through a break in the cecal mucosa. Of secondary concern will be the more common acute pericecitis, caused by an inflamed appendix, fallopian tube, or other organ lying against the cecum and involving it *contiguously*.

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TABLE I

Outline of 23 cases of cecal inflammations

	No. Cases
I. Acute primary (idiopathic) cecitis	7
II. Acute secondary cecitis	8
A. To appendicitis	4
B. To ileitis	2
C. To diverticulitis	1
D. Following appendectomy	1
III. Acute secondary pericecitis	6
A. To salpingitis	2
B. To appendicitis	1
C. To diverticulitis	1
D. To perforation of ileum	1
E. Uncertain origin	1
IV. Perforations of the cecum	2
A. Due to fecolith	1
B. Uncertain origin	1
Total	23

One final point concerning terminology is noteworthy. The upper boundary of the cecum as viewed by the surgeon is rather generous, including all of that allowed by the anatomist—up to the level of the ileal opening—and perhaps an inch or so more. The more generous definition will be used in this report.

MATERIAL

Table I outlines the 23 cases which form the basis for this study. The 7 instances of primary (idiopathic) cecitis represent the total number of this type which have been recorded at Charity Hospital in New Orleans over a 15 year period (1937 to 1952). The remaining 16 cases were selected at random to illustrate the various methods by which the cecum may become inflamed.

The entire group of cases is classified for convenience into four major categories:

1. Acute Primary (Idiopathic) Cecitis (table II)
2. Acute Secondary Cecitis (table III)
3. Acute Secondary Pericecitis (table IV)
4. Acute Perforations of the Cecum (table V)

INCIDENCE

Incidence of Primary Type. The existence of the primary type of cecitis is denied by some authors[7, 10, 13] who maintain that a diseased or previously diseased appendix is the underlying cause. It seems reasonable that if one recognizes cases of nonobstructive appendicitis as being *primary*, then one might also admit the same possibility in the cecum.

Instances of cecitis of the primary type were reviewed in 1943 by Spivack and Busch[21] who collected 35 cases from the literature and added 2 of their own.

TABLE II

*Seven cases of primary (idiopathic) cecitis**

Case No., Initials	Age, Sex	Length of Illness	Operative Findings†	Procedure	Pathology Report	Miscellaneous
1 C.A.	45 ♂	3 days	3 reddened and edematous areas (2 by 3 cm. each) in ant. wall of cecum	Biopsy of cecum Appendectomy	Acute cecitis Normal appendix	Re-explored two months later; cecum normal
2 W.S.	32 ♂	4 days	2 by 3 cm. friable & reddened area in caput of cecum; carcinoma suspected	Ileocolic resection	Acute cecitis; 1.5 cm. intramural abscess; ileum & appendix normal	Uneventful recovery
3 W.C.	27 ♂	½ day	4 by 4 cm. area of edema, redness & induration in anterolateral cecum	Biopsy of cecum Appendectomy	Acute cecitis Normal appendix	Re-explored one month later; cecitis subsiding
4 M.B.	25 ♀	4 days	3.5 cm. circular area of acute inflammation in anterior cecal wall	Biopsy of cecum Appendectomy	Biopsy specimen lost Normal appendix	Uneventful recovery
5 A.D.	37 ♀	1 day	2.5 by 4.5 cm. indurated & edematous area in lateral wall of cecum	Appendectomy	Normal appendix	Uneventful recovery
6 P.W.	20 ♂	1 day	Entire cecum diffusely thickened & granular with fibrinous exudate; ileum & appendix normal	None	—	Uneventful recovery
7 J.McK.	51 ♂	1 day	Indurated, red. & edematous area in anterolateral cecum (size not stated)	Biopsy of cecum Appendectomy Inversion of area of cecitis	Acute cecitis Fibrosis of appendix	Stools positive for strongloides Uneventful recovery

* No patient in this group had a palpable mass on admission.

† Unless otherwise stated, appendix and terminal ileum normal at surgery.

TABLE III

Eight cases of acute secondary cecitis

Case No., Initials	Age, Sex	Length of Illness	Operative Findings*	Procedure	Pathology Report	Miscellaneous
8 E.B.S.	65 ♀	7 days	6 cm. area of induration, edema & thickening in anterolateral wall of cecum Appendix grossly normal; Few enlarged lymph nodes in ileocolic region	Biopsy of cecum Appendectomy Biopsy of lymph node	Acute suppurative cecitis Acute appendicitis (nonobstructive) Acute adenitis	† Uneventful recovery
9 C.T.	16 ♂	2 weeks	Entire cecum anteriorly thick and inflamed; local peritonitis present; early acute appendicitis (not in contact with cecum)	Culture of exudate Appendectomy	Culture: Shigella organisms Acute focal appendicitis	Uneventful recovery
10 J.W.	15 ♀	1 day	Acute appendicitis (not lying against cecum); area of acute cecitis around base of appendix; small abscess in wall of cecum	Appendectomy Drainage of abscess into lumen of cecum	Acute appendicitis (nonobstructive)	Uneventful recovery
11 S.C.	53 ♂	1½ days	Acute obstructive appendicitis; 6 cm. area of acute cecitis around base of appendix (See fig. 2)	Appendectomy Cecostomy	Acute suppurative appendicitis	Uneventful recovery
12 O.P.	18 ♀	3 weeks	Distal ⅓ of appendix edematous; entire cecum red, indurated & edematous. Distal 20 cm. of ileum acutely inflamed	Appendectomy	Acute periappendicitis	Uneventful recovery

13 E.B.	38 ♂	Recurrent, 4 weeks; worse, two days	Terminal 15 cms of ileum & ileocecal junction red, friable, & indurated. Acutely inflamed cecum around ileal opening	Ileocolic resection	Acute regional ileocolitis; Normal appendix	Postop. wound infection; uneventful recovery

Case 14. J.W., 37 year old man, was hospitalized for 11 days in 1943 for possible appendicitis, but no diagnosis reached. Rehospitalized in December 1948, for abscess in right lower abdominal quadrant of 3 weeks' duration. Treatment was conservative. Readmitted in February 1949, for interval appendectomy. At surgery, appendix adherent to cecum by old adhesions, and a thick and indurated mass was present in lateral wall of cecum. Appendectomy done. Pathology report was fibrosis of submucosa of appendix. Readmitted in October 1949, and had drainage of another abscess in right lower abdomen. Uneventful recovery. Barium enema in November 1949, showed cecal diverticulosis and a mass in cecum. Rehospitalized in December 1949, for drainage of another abdominal abscess. Developed external cecal fistula two weeks later. Drainage gradually ceased. Readmitted in March 1950, and a right hemicolectomy done. Uneventful recovery. Pathology report was cecal diverticulosis (12 or more diverticula), diverticulitis, and chronic cecitis. Readmitted and reexplored in April 1950, for intestinal obstruction due to adhesions. Uneventful recovery. At last follow-up visit in May 1952, was asymptomatic.

Case 15. A 19 year old female, was admitted with abdominal pain of 3 weeks' duration. An appendectomy had been done elsewhere two weeks before onset of pain. Examination disclosed a palpable and tender mass in the right lower abdomen. Preoperative diagnosis was abscess in abdominal wall versus postappendiceal intra-abdominal abscess. At surgery on day of admission the entire cecum was found to be edematous, indurated and thickened. The appendectomy site was well healed. Terminal ileum was normal. No enlarged mesenteric nodes present. Biopsy specimen of cecum taken. Pathology report was acute and chronic inflammation of cecum with marked edema. Postoperative barium enema showed irregularity of cecal mucosa. Uneventful recovery. Discharged on twentieth postoperative day. No follow-up.

* Unless otherwise stated, ileum and appendix normal at surgery.

† Palpable mass in right lower abdomen on admission.

TABLE IV

Six cases of acute secondary pericecitis

Case No., Initials	Age, Sex	Length of Illness	Operative Findings*	Procedure	Pathology Report	Miscellaneous
16 H.H.	19 ♀	3 days	Acute salpingitis; caput of cecum in pelvis and adherent to right tube; cecum acutely inflamed, granular	Appendectomy	Normal appendix	Uneventful recovery
17 W.H.	26 ♀	2 days	Both tubes and ovaries acutely inflamed; cecum in pelvis and lying against right tube; 3.5 cm. area of edema, redness, and thickening in caput of cecum	Appendectomy	Acute periappendicitis	Uneventful recovery
18 R.S.	36 ♂	8 days	Acute retrocecal appendicitis with abscess; marked edema and thickening of overlying cecum	Appendectomy Cecostomy	Acute perforative appendicitis	Readmitted 4 weeks later for intestinal obstruction. Recovery
19 L.B.	56 ♀	½ day	Acutely inflamed diverticulum, lateral wall of cecum. Surrounding cecum edematous and red with fibrinous exudate	Excision of diverticulum Biopsy of cecum Appendectomy Cecostomy	Acute suppurative diverticulitis Acute cecitis Normal appendix	Uneventful recovery
20 C.H.	29 ♂	9 days	Generalized peritonitis; terminal ileum plastered against cecum; one cm. perforation of ileum; cecum acutely inflamed	Closure of ileal perforation Culture of exudate	Culture: typhoid organisms	Stormy postop. course. Recovery
21 D.S.	13 ♀	9 days	Appendix lying against cecum; both thickened, red, and indurated with fresh exudate. Area of cecitis 5 cm. in diameter	Appendectomy Exteriorization of cecum Cecostomy	Subacute periappendicitis Pericecal abscess with refractile bodies (cellulose?)	Uneventful recovery

* Unless otherwise stated, terminal ileum and appendix normal at surgery.

(cellulose?)

* Unless otherwise stated, terminal ileum and appendix normal at surgery.

TABLE V

Two cases of acute perforations of the cecum

Case No., Initials	Age, Sex	Length of Illness	Operative Findings*	Procedure	Pathology Report	Miscellaneous
22 F.St.M.	44 ♂	4 weeks	Abscess with 50 cc. pus present on lateral side of cecum. 1 cm. perforation of cecum found; small intramural abscess also present near perforation	Closure of cecal perforation Pericecal drainage	—	Readmitted 5 months later; I & D of RLQ abscess. Recovery. Barium enema one yr. later normal
23 M.B.	43 ♀	3 days	Generalized purulent peritonitis; 2.5 cm. hard fecolith lying outside cecum and overlying 2.5 cm. ragged perforation of cecum on its lateral aspect. Cecum around perforation acutely inflamed	Biopsy of cecum Closure of cecal perforation	Acute cecitis Fecalith	Stormy postoperative course. Recovery

* Unless otherwise stated, terminal ileum and appendix normal at surgery.

Since that time approximately 7 other cases have been recorded.[4, 12, 15, 16, 18] Eleven additional cases have been reported in which an inflammation of the cecum spread to involve some or all of the right colon.[22]

Incidence of Secondary Type. Cecitis secondary to appendicitis appears to be uncommon if the number of cases reported is used as an index. Acute appendicitis is believed to be the most common cause of acute secondary cecitis at this hospital, and it is surprising that this type has received only scant attention in the literature. Moreover, of the few case reports available, the majority are concerned with a suppurative or perforative appendicitis lying against the cecum and involving its serosa first. As such they represent examples of pericecitis as shown in figure 4.

Cecitis incidental to perforations of the cecum by ingested foreign bodies were tabulated by Macmanus[14] in 1941. In this review of 95 perforations of the intestinal tract by foreign bodies, 73 per cent were found in the ileocecal region, 11 involving the cecum alone and producing a surrounding zone of acute cecitis or pericecitis.

A substantial number of the cases of cecal diverticulitis have an associated cecitis or pericecitis of variable degree. In some instances this secondary reaction may be more alarming than the underlying condition.

Various estimates[2] indicate that in about 30 per cent of the patients with regional ileitis the process is of an acute nature when first seen, and that the cecum is secondarily invaded in approximately one-third of all cases, particularly in the subacute or chronic stages.

Incidence of Pericecitis. No figures are available in the English literature for an appraisal of the incidence of this type. On the basis of personal experience, unsupported by factual data, the two most common causes of acute pericecitis are complications of appendicitis, in which the appendix lies adjacent to the cecum, and acute salpingitis, in which the cecum or the fallopian tube is mobile enough to allow direct contact.

PATHOGENESIS

Pathogenesis of Primary Cecitis. The offending organism(s) must, in some manner, reach the rich supply of lymphatic vessels and solitary nodules in the submucosa of the cecum in order to initiate a chain of events. Minute abrasions of the cecal mucosa by foreign bodies, parasites, and hard feces are stressed by several authors[20] as being of importance in permitting organisms to enter the wall of the cecum. This explanation is identical in theory with that of appendicitis of the nonobstructive type.

Pathogenesis of Secondary Cecitis. No satisfactory mechanism has as yet been proposed to explain this type of cecitis. Any proposal must explain why cecal involvement frequently is seen in terminal ileitis, a relatively uncommon disease, and why it is seen less frequently in appendicitis, a common disease.

There are no anatomic barriers to prevent such a spread, inasmuch as the submucosa of the cecum is continuous with that of the appendix, terminal ileum, and ascending colon.[8] Examples of this intramural spread from the appendix

are seen in table III. In all 3 cases the appendicitis was minimal in degree and of the nonobstructive type, and the cecum and appendix were in contact only at their usual attachment. There is little reason to doubt that in these instances the avenue of spread was from the wall of one structure to the wall of the other.

It is well known that most cases of appendicitis are of the obstructive type and that a reasonably healthy segment of appendix usually is present between the cecum and the site of obstruction (fig. 1). It is believed that it is this segment of appendix, however small it may be, which affords the cecum a protective barrier. Were it not for this obstructive element in appendicitis, it is probable that acute secondary cecitis would be a more common disease.

The proposal that appendiceal obstruction plays an important role in preventing cecitis is supported by the operative findings in case 11 (fig. 2). In this particular instance the cecum became involved despite the presence of an obstructing fecalith. The unusual location of the fecalith in the intramural portion of the appendix allowed no room for a protective barrier to develop.

In addition to fecaloid concretions, any factor such as enlarged lymph follicles, pronounced edema, ingested foreign bodies, or parasites equally may be as effective in producing a submucosal blockade in appendicitis.

It is antithetical to state that appendiceal obstruction is desirable on one hand in preventing cecitis, and that it is undesirable on the other in causing a potentially dangerous closed loop obstruction of the appendix. Nevertheless, this represents an accurate statement of beliefs.

In contrast, there is no similar obstructive factor present in terminal ileitis, and cecal involvement is notoriously common, especially in the established cases. It is not unreasonable to suggest that the mechanism whereby cecitis occurs

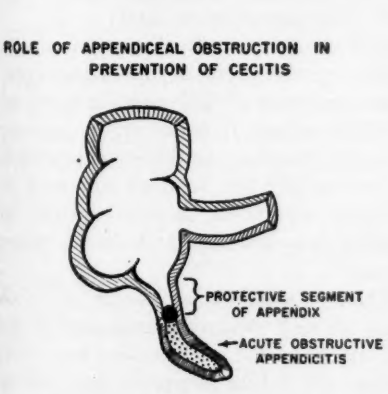
ROLE OF APPENDICEAL OBSTRUCTION IN PREVENTION OF CECITIS
PROTECTIVE SEGMENT OF APPENDIX
ACUTE OBSTRUCTIVE APPENDICITIS

FIG. 1

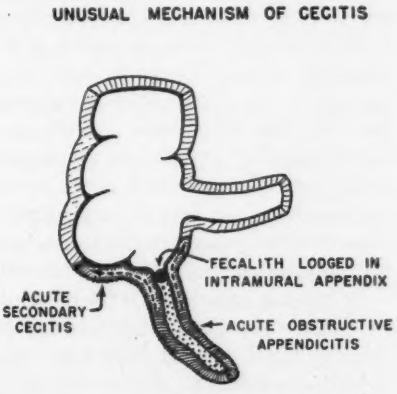

FIG. 2

FIG. 1. Mechanism whereby segment of healthy appendix tends to limit proximal spread of inflammation from appendicitis. Obstructing factors may be fecaliths, enlarged lymph follicles, marked edema of appendiceal wall, parasites, or ingested foreign bodies.

FIG. 2. Mechanism of acute cecitis in case 11. Unusual location of fecalith in intramural portion of appendix allowed no room for protective barrier to develop. A 6 cm. area of acute cecitis was present around base of appendix.

CAUSES OF ACUTE SECONDARY CECITIS

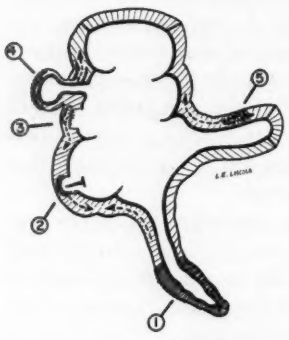

FIG. 3

CAUSES OF ACUTE PERICECITIS

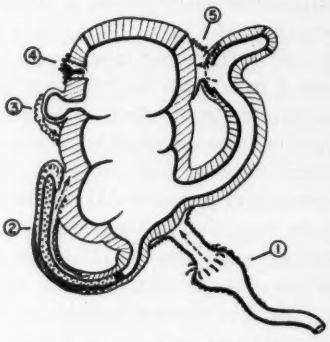

FIG. 4

FIG. 3. 1. Acute appendicitis (cases 8, 9, 10, 11), 2. Foreign body perforations (see ref. 14), 3. Specific or nonspecific ulcerations (case 22), 4. Acute diverticulitis (case 14), 5. Terminal ileitis (cases 12, 13).

FIG. 4. 1. Acute salpingitis (cases 16, 17), 2. Acute appendicitis (case 18), 3. Acute diverticulitis (case 19), 4. Perforations of cecum (case 23), 5. Perforative diseases of terminal ileum (case 20).

secondary to ileitis or to appendicitis may well be identical, the only difference being the nature of the infecting agent.

A fairly wide variety of other local conditions besides appendicitis and ileitis may result in an acute cecitis (fig. 3). Diverticulitis, specific or nonspecific ulcerations of the cecal mucosa, and foreign body perforations may all furnish the necessary break in mucosal continuity and allow organisms to enter.

Mechanism in Acute Pericecitis. A distinctly different route of invasion occurs when an inflamed appendix, fallopian tube, or other structure lies against the cecum (fig. 4). Under these circumstances the serosa of the cecum is involved first and this layer tends to limit any further spread. If, however, the inflammatory process adjacent to the cecum is quite purulent or the serosal contact prolonged, the entire wall of the cecum may be affected. In some advanced or neglected cases it may be impossible, or even impractical, to state whether an inflammatory process in the cecum is primary or secondary, or whether it began on the inside or the outside of this structure.

Of passing importance is the recognition that tuberculosis, amebiasis, ileocolitis of Crohn's type, actinomycosis and others all may produce granulomas in the cecum. These specific infections usually are attended with more chronicity, systemic complaints, obstruction, ulceration, and fistula formation than will be found in acute cecitis. In none of the 23 patients in this group were these diseases mentioned in the differential diagnosis.

PATHOLOGY

Of all layers of the cecum, the submucosa contains the largest blood vessels and most lymph channels and solitary nodules embedded in a loose connective tissue

stroma. It is not surprising that this layer reacts more violently in response to bacterial invasion. The early edema and thickening of the submucosal coat is a defense mechanism which characterizes not only the cecum, but the entire gastrointestinal tract as well.

Whether the basic pathologic process is a submucosal cellulitis or lymphangitis, or both, is not certain. The patchy distribution of the earlier lesions (case 1), in which the involved areas are sharply demarcated from the uninvolved portions of the cecum, is difficult to explain other than on a lymphangitic basis. In the other cases such skip areas were absent and the process resembled that of a spreading cellulitis.

For many years it has been customary to describe two gross forms of cecitis, the circumscribed and the diffuse. In this group of 23 cases only 5 could be so categorized, 4 being the diffuse form and 1 the circumscribed. The remaining cases, with the exception of the 2 cecal perforations, presented a fairly well localized, reddened, edematous, and indurated area in the wall of the cecum. The classification of cecitis into these two gross forms proved to be of no practical value in this study.

Cecitis of all types (primary, secondary, and pericecitis) tends to subside spontaneously in the majority of cases, and this was noted in every patient in this study in whom the lesion was observed and not resected. Occasionally small abscesses may form in the wall of the cecum, and this was noted in 4 patients. In 2 of these, foreign bodies resembling cellulose fibers were found within the abscess cavities. Their significance is speculative.

Frank perforation of the cecum was present in 2 patients (table V), and in both of these the perforation appeared to be a primary event and not a complication of a pre-existing cecitis. In 1 instance the perforation apparently was caused by a hard fecalith 1 inch in diameter which was present on the outside of the cecum and which was overlying a 1 inch ragged perforation. A similar case was recently reported by Blotner.[3] The entrapping of a fecalith in a cecal haustration of about the same size, followed by pressure necrosis, is the accepted explanation, however unappealing it might be.

If complete resolution of an inflamed cecum does not occur, it is possible that

TABLE VI

Length of follow-up in 20 cases of cecitis not resected

Length of Follow-up	*No. Cases*
A. None	2
B. Months:	
1 to 3	4
4 to 8	3
9 to 11	3
C. Years:	
1 to 3	4
4 to 8	2
9 to 11	2
Total	20

the process could become chronic with the formation of an inflammatory tumor. In none of the 18 patients in this study who were followed (table VI) did a granuloma develop.

As a final remark on the pathology of cecitis, it is well to remember that the surgeon's description of a cecal inflammation is hampered in that it is not advisable to open the cecum in situ in order to examine its interior. Palpation being seriously handicapped by edema, friability, and induration, the only reliable observation which remains is the external appearance of such a lesion. To examine and describe only one side of a specimen, particularly when the underlying pathology well may be on the other side, is, at most, a unilateral approach to gross pathology. Biopsy specimens of the cecum, generally including only its outer layers, are similarly unreliable.

Microscopic Pathology. Material for microscopic study of the cecum was available in 11 patients, 8 being biopsy specimens. Nothing characteristic about the histologic picture can be stated to occur, and all stages of nonspecific inflammation were noted from early and pronounced edema to the later stages of suppuration and formation of intramural abscesses. Of all layers of the cecum, the submucosa was seen consistently to suffer the most.

DIAGNOSIS

Fortunately the peritoneal signs of cecal inflammations usually give sufficient indications to open the abdomen. In 22 of the 23 patients in this study the preoperative diagnosis was acute appendicitis or a complication of appendicitis (table VII).

Similar difficulties in diagnosis are mentioned in the literature. The presence of a palpable and tender mass in the right lower abdominal quadrant within 48 hours after the onset of symptoms is supposedly suggestive of cecitis, but in only 3 patients in this group was a mass noted. No reliable clues were found in this study by which cecitis could be detected prior to operation. The symptoms, physical findings, leukocyte count, and degree of fever were indistinguishable from those of appendicitis in its various stages (table VIII).

Even with the abdomen open and the cecal lesion in view the diagnosis often may be difficult. The primary type of cecitis is diagnosed by exclusion. The

TABLE VII

Preoperative diagnoses in 23 cases of cecitis (all types)

	No. Cases
1. Acute appendicitis	15
2. Appendiceal abscess	2
3. Appendicitis with local peritonitis	2
4. Appendicitis with general peritonitis	1
5. Appendicitis versus salpingitis	1
6. Postappendectomy abscess	1
7. Perforation of terminal ileum	1
Total	23

TABLE VIII

*Summary of clinical features in 22 instances of cecitis and pericecitis**

Physical Findings—No. Cases (Abdominal)	Admission Temperature—No. Cases (Degrees F.).	White Cell Count—No. Cases
1. RLQ tenderness—5	Normal—1	Under 8,000—4
2. RLQ tenderness and rebound tenderness—9	99 to 100—13	8,000 to 11,000—4
3. RLQ rebound tenderness and rigidity—4	100.2 to 101—5	11,000 to 13,000—5
4. Tender RLQ mass—2	101.2 to 102—2	13,000 to 16,000—6
5. Signs of generalized peritonitis—2	102.2 to 103—1	16,000 to 18,000—1
		18,000 to 22,000—2

*Case 14 omitted from this summary because of numerous hospital admissions.

secondary type may be related to a variety of coexistent diseases such as appendicitis, diverticulitis, ileitis, ulcerations of the cecal mucosa, and foreign body perforations.

The recognition of pericecitis is dependent upon the finding of an inflamed organ, such as the appendix or fallopian tube, lying against the cecum. As a general rule this is not difficult except when abscess formation obscures the associated or underlying pathologic process.

MANAGEMENT

Of foremost consideration in the management of these cases is the awareness that such an entity as cecitis exists. In many instances it is likely that the true nature of the condition may remain unrecognized even at operation.

Of the 44 cases of primary cecitis in the literature, 13 patients had radical ileocolic resections, 9 had local resections, and 20 had appendectomies with or without biopsy of the cecum. In most of the resected cases it appeared that a suspicious granulomatous tumor was the basis for its removal; in others, the inflammatory mass must have appeared too bulky or threatening to be left within the abdomen.

The surgical procedures used in this study are listed in table IX. Only three lesions were resected, and in 1 of these the necessity for resection could be seriously questioned. This conservatism in management is in striking contrast to the high resection rate recorded in the older literature.

There is general agreement that in uncomplicated cases of primary cecitis the procedure of choice is not to disturb the cecum except to take a biopsy specimen. If the disease in the cecum is secondary to appendicitis, salpingitis, or diverticulitis, the associated pathologic process can be managed, with but few exceptions, according to accepted standards as if the cecum were not involved.

Generally, the local complications of cecitis most likely to be of concern to the surgeon may be listed as follows, together with a brief plan of management:

1. *Abscess Formation (Intramural or Pericecal).* (Cases 10, 14, 18). Incision and drainage of all pericecal abscesses is mandatory, employing an extraperitoneal approach if at all feasible. Small intramural collections of pus, usually represent-

TABLE IX

Surgical procedures used in 23 cases of cecitis

Procedure	*No. Cases*
1. Appendectomy	14
2. Biopsy of cecum	9
3. Cecostomy	3
4. Ileocolic resection	3
5. Drainage of abscesses	3
6. Closure of perforation of cecum	2
7. Exteriorization of cecum	1
8. Inversion of area of cecitis	1
9. Excision of cecal diverticulum	1
10. Closure of perforation of ileum	1
11. Biopsy of ileocolic lymph node	1
12. None	1

ing only 1 to 2 cc. of purulent matter, may be left alone, aspirated, or drained externally. If the latter method is used, a tube cecostomy at the drainage site should be considered.

2. *Frank or Imminent Perforation of Cecum* (Cases 22, 23). Occasionally the smaller perforations may be successfully closed by sutures and the area surrounding the cecum drained liberally. More commonly, some type of cecostomy will be found advisable. More extensive defects may require exteriorization procedures.

3. *Obstruction at Ileocecal Opening* (Case 13). This may be secondary to edema or inflammation, or to larger collections of pus in the wall of the cecum adjacent to the ileal opening, and may warrant a short-circuiting procedure or ileocolic resection, depending upon the extent and nature of the pathology encountered.

4. *Edematous and Friable Cecum* (Cases 11, 18, 21). If, following appendectomy, the edematous and friable cecum will not anchor an inverting suture for the base of the appendix, a cecostomy of some type is advisable.

5. *Carcinoma Versus Inflammatory Tumor* (Cases 1, 2, 3). In such situations, refinements in surgical judgement are extremely helpful. As these lesions are almost always unsuspected before operation, the bowel contents are not prepared, and under such circumstances it is *safest* to close the abdomen and prepare the patient and the bowel for subsequent re-exploration. Advantage of this period of preparation may be taken to administer a course of antibiotic drugs. This plan of management was utilized in 2 patients in this group (table II). In both patients, after a course of antibiotics was given, a defect of the cecum persisted on repeated barium enema studies. On re-exploration the inflammatory process had subsided and neither patient required resection.

MISCELLANEOUS OBSERVATIONS

Results of Barium Enema Studies. Postoperative barium enemas were obtained in 8 patients. On the basis of these roentgenographic findings, it can be stated

that deformities of the cecum may persist on the roentgenogram for 4 to 6 weeks and occasionally longer. Despite the deformities on the roentgenogram, all of the patients were noted to be without symptoms during this period of time.

Length of Hospital Stay. The average length of hospitalization for the entire group was 17 days. Of the 14 cases which occurred after the advent of chemotherapeutic and antibiotic drugs, the average period of hospital stay was about nine days.

Mortality Rate. There were no deaths in this group of 23 patients. The often quoted mortality rate for primary cecitis is about 10 per cent.[4] However this figure was determined in a group of patients in whom the rate of resection was excessively high and in a period prior to the introduction of antibiotic drugs.

SUMMARY

In a study of 23 instances of acute cecal inflammations, 7 were presented as representing the primary (idiopathic) type. The remaining 16 cases were random selections to illustrate the various mechanisms by which the cecum could become secondarily inflamed.

The clinical features of these inflammations were found to be indistinguishable from those of appendicitis in its various stages.

An effort was made to correlate certain details of the pathogenesis and pathologic changes of these lesions, emphasizing that our current understanding of this subject is handicapped by too many variations in terminology.

A standardized classification for these disorders was proposed, and a plea made that the terms *typhlitis* and *phlegmon* be retired from active use in the medical literature.

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A NEW METHOD OF MANAGEMENT FOR THROMBOSIS OF DEEP VEINS OF THE EXTREMITIES

THROMBECTOMY, RESTORATION OF THE LUMEN AND HEPARINIZATION

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No single method of treatment for deep vein thrombosis has proved entirely satisfactory. Evidences of this are the numerous measures, many with violent proponents, none of which has provided outstanding superiority. Herein is described a method of management emanating from a different conception and new aim of attainment, namely to get the lumen of the vein open and to keep it open.

As soon as the diagnosis of deep vein thrombosis is made, if the clot involves a large vein, operation is decided upon and the femoral or iliac or the axillary or subclavian is exposed at a level which would be judged to be near the upper segment of the clot. A longitudinal incision is made into the thrombosed vein and the clot is removed (fig. 1). If the thrombosis is an early one this is not difficult. The clot will sinuously work out of the opening in the vein or it can be coaxed out by gently pulling it with forceps or it can be expressed by downward pressure along the course of the vein from above or upward pressure along the course of the vein beginning some distance below the incision (fig. 2). When the clot has been removed completely from a long segment below or above the incision in the vein, furious bleeding will take place which has to be controlled by two soft rubber strips previously placed around that segment of the vein. Sometimes of value in removing the clot are long slender forceps inserted into the vein, with which the clot is caught and pulled out; or the application of suction either by a rigid suction tube or by a small (no. 12 F) blunt end soft rubber catheter inserted into the vein lumen. When the tube is introduced into the vein it is alternately advanced and withdrawn carefully until the clot is removed and free bleeding is encountered.

Removal of a clot from a thrombosed deep vein has been done, many times of course, particularly by surgeons who have ligated the veins with the intention of producing a favorable result and preventing pulmonary embolism. The next step in our routine however is different. Instead of ligating the vein, we close it leaving the lumen open. The incision in the vein is closed with no. 0000 silk using a running Carrel type mattress suture. The lumen of the vein is thus not obstructed. The incision in the fascia and skin is closed. Then the saphenous vein at the ankle is exposed in the same leg through a longitudinal incision just above and on the anterolateral aspect of the internal malleolus. A no. PE 100 polythene tube is used to intubate the proximal segment, and the distal segment is ligated (fig. 3). The polythene tubing is then connected by a needle to an infusion of 5 per cent dextrose. A continuous drip is established at the rate of about 20 drops a minute.

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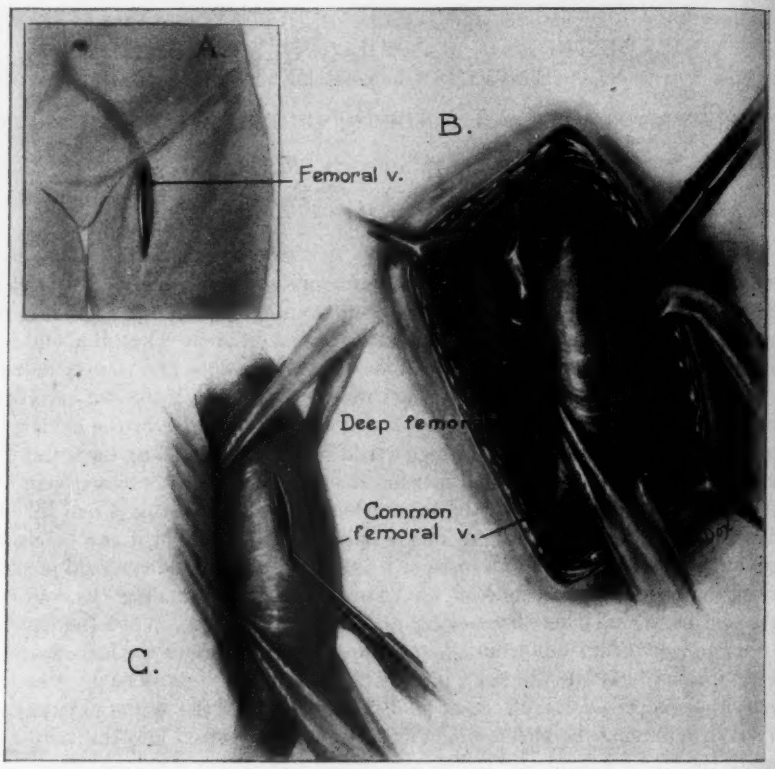

FIG. 1. (a) Incision is made over the deep vein at a level which is expected to be near the upper end of the clot. (b) After isolating the thrombosed vein, small rubber tissue drains are placed beneath it. (c) A longitudinal incision is made in the vein.

At this rate infusion of 1000 cc. will run almost 12 hours. One hour after the operation is completed 200 mg. of heparin are added to the 1000 cc. flask of 5 per cent dextrose. This interval of 1 hour is arbitrarily established to give time for sealing of capillary and suture lines. When the heparin was added immediately oozing occurred into the wound and a hematoma resulted. The clotting time is checked every 4 hours by the Lee-White method. An attempt is made to maintain a coagulation time of 15 to 20 minutes. Near the end of the first 12 hour period, when the 1000 cc. of infusion is almost complete, another 1000 cc. of 5 per cent dextrose is added. From 100 to 200 mg. of heparin are dissolved in this 1000 cc. depending on the variation of the clotting time from normal. Additional 50 mg. or 100 mg. doses of heparin may be added during the time infusion is running if this is necessary. An additional control on the clotting time is the rate of infusion. It may be speeded up or slowed down.

In addition to this treatment the nurse is instructed to put a tourniquet around the leg just above the knee, sufficiently tight to obstruct the flow in the super-

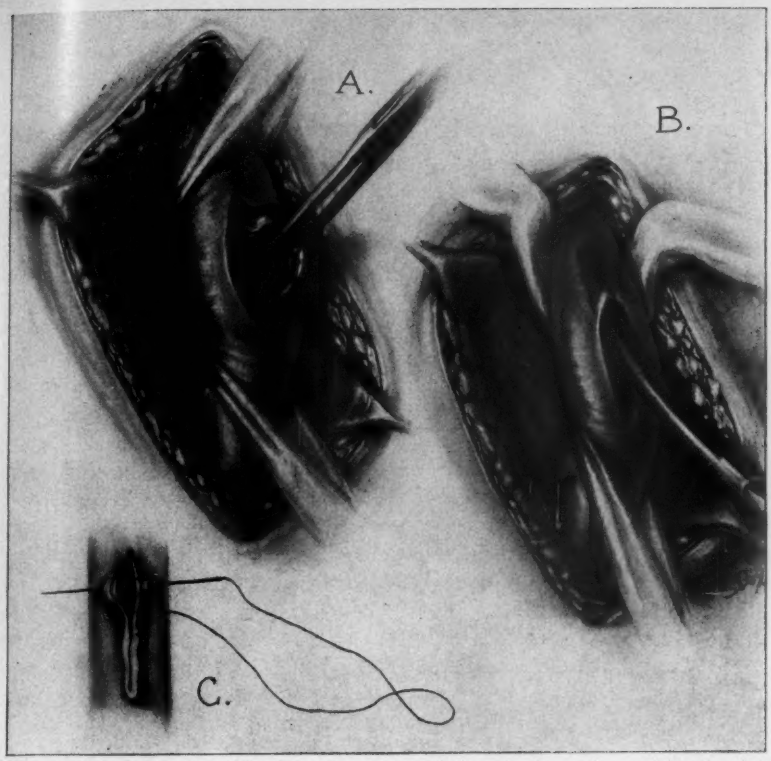

FIG. 2. Compression is made along the course of the vein from above while the clot is gently lifted out, with an instrument. Then pressure from below upward is exerted to remove the clot from the lower segment. If the flow of blood is free from each direction it indicates that the clot has been adequately removed. (b) If blood flow is not normal a catheter, with suction attached, is introduced in each segment in order to remove adherent thrombus. (c) The vein is not ligated but the lumen is restored by suturing the opening with no. 0000 silk.

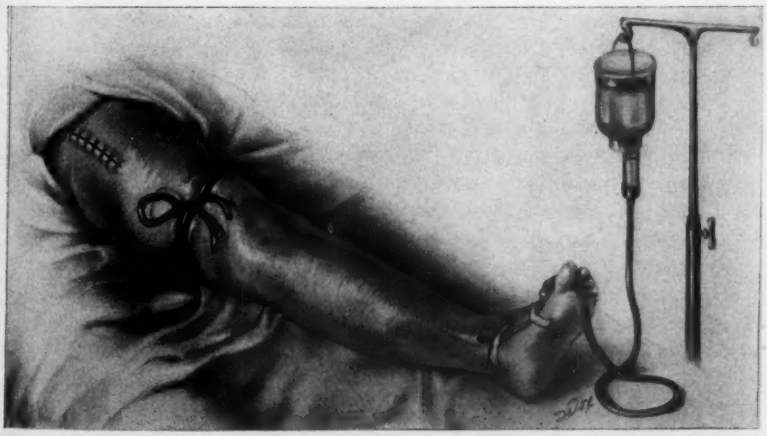

FIG. 3. At the end of operation a polythene tube is inserted into the saphenous vein at the ankle and a solution containing heparin, (approximately 200 mg. to 1000 cc.) is dripped very slowly. Each hour for 5 to 10 minutes a tourniquet is applied above the knee sufficiently tight to constrict the superficial circulation and shunt the flow of heparin more definitely through the deep segment of vein from which the thrombus has been removed. Although the infusion is started immediately it is advisable to delay the addition of heparin for a period of one hour after operation in order to permit sealing of suture lines.

ficial circulation for 5 minutes every hour during the first 12 hours. A more accurate way is to pump up a blood pressure cuff applied just above the knee to 35 mm. of mercury. This more definitely shunts the heparin solution into the deep system. Probably adequate heparinization occurs even without the use of a tourniquet. The heparin solution is continued without interruption for a period of 72 hours and then is stopped. Then the patient is permitted to be ambulatory for brief periods of time with supporting elastic bandages.

This method has been used by us in 6 patients and the results have been brilliant when the deep vein thromboses have been of short duration, even though the clot was extensive and the swelling massive.

This type of therapy is pre-eminently indicated in extensive venous thrombosis with massive edema giving the picture of phlegmasia alba dolens or phlegmasia cerulia dolens.[1] Gangrene associated with and caused by massive thrombosis of deep veins is being reported with increasing frequency. The first report of this condition was made by Gregoire and associates in 1938.[3] Haimovici,[4] in 1950, collected 28 cases from the literature which included 2 of his own. Forty-four per cent of the cases of gangrene occurred in phlegmasia alba dolens and 56 per cent in phlegmasia cerulia dolens. Periarterial sympathectomy has failed to stop the gangrene according to Decoulx and Bastien.[2] Veal,[5] who reported 11 cases of acute massive venous occlusion, 2 with gangrene, believes that interruption of the sympathetic pathways by nerve blocks may actually increase the vascular congestion. It is not unlikely that the method described herein would prevent such a complication. Massive venous occlusion was encountered in 4 of the 6 cases reported herein. The subsidence of the edema after the removal of the clot was prompt and dramatic.

The time such thrombectomy can be effective is dependent upon the condition of the clot and whether it actually can be removed. Surprisingly, even weeks after the initiation of the process, a long segment of the clot in a femoral vein still may be in the *red* state and organization may not be complete at any level or it may be complete in a small (e.g. 1 cm.) segment while unorganized clot many times longer exists on either side of it. It would seem that the extent of thrombosis, i.e. the length of the segment, of the vein involved is a big factor in the degree of edema in the extremity. When the thrombosed segment of a deep vein is short it is conjectural whether *run around* collaterals will protect the limb against massive edema; but it is obvious that when a long and high segment is involved massive edema results. In the cases herein reported it has been amazing to us how extensive and how long the clots are.

Probably very little edema is seen in the patients in whom the thrombosis is only in very small veins, such as veins of the calf of the leg. When evidence suggests that this alone is the case, the operation and type of management described herein has not been used nor advocated. On the contrary such cases have been managed conservatively with anticoagulants only, until they recovered or until they progressed into a more advanced stage of thrombosis when the more aggressive management was instituted (see case 6).

Among the cases reported is one of effort thrombosis involving the subclavian

vein with massive edema of the upper extremity. Removing the clot gave a prompt and brilliant result. Immediately after operation, the edema of the arm receded. When the incision was first made into the vein, bleeding from the distal segment was profuse and under great pressure. The obstruction proximal to this was sucked out with a catheter.

The rationale of removing the clot and closing the opening in the major vein is that it attempts to open and keep open a major channel for the return circulation. If this can be accomplished it will tremendously reduce morbidity, the post-thrombophlebetic edema and all its sequelae. The objection that the valves are destroyed by the clot and that an untied vein is not desirable may be countered by the fact that most proponents of ligating the femoral vein for post-thrombophlebetic edema have given it up after finding its results undesirable. Moreover, after removing an extensive but recent thrombosis, we have found valves intact as evidenced by their obstruction to the passage of a catheter into the distal segment even though on removal of the clot a profuse flow of blood resulted. Thus up to a certain time, at least, removing the clot may preserve the valves.

Thrombosis during pregnancy and the postpartum state proposes a complex problem. A 22 year old woman, who developed deep vein thrombosis during a period when her obstetrician kept her in bed to combat a threatened abortion, was seen during the period of these studies. The onset of deep vein thrombosis was insidious. When we were called to see her after she had been in bed for eight days her left leg and thigh was moderately swollen. We hesitated to use anticoagulants because of the bleeding, and, because of that decision, we would not advise active intervention and removal of the clot. In retrospect the prolonged and persistent post-thrombophlebetic edema, now present six months under conservative treatment suggests that it might have been better had the clot been removed and the patient been gradually activated by ambulation, even though anticoagulants could not have been advised under the circumstances. Postpartum thrombosis may prove just as difficult. Removing the clot and closing the vein may be far better than not removing it or removing it and ligating the vein. The next time I have the opportunity I intend to try removing the clot and closing the vein even though anticoagulants may not be employed.

There is no question that ligation of veins increases the pressure in the venous system distal to that level. My own personal experience and viewpoint is that never is a deep vein ligated with impunity. There is always residual edema. It may be latent because the patient is protecting the extremity by rest and restricted activity, but if there is long dependency of the extremity during the day it becomes manifest. In some instances it is well indicated and even absolutely necessary to ligate deep veins and not use excision of a thrombus and closure of a vein without ligation. The indications for ligation, I believe, are few but they are absolute when there is septic thrombosis. In instances of pelvic thrombosis with septicemia, ligation of the ovarian veins and inferior vena cava should be done and it may be lifesaving. A second but less absolute indication exists when there has been mild pulmonary embolism. Ligation may be done to prevent a major embolus and death. However, at present it appears to me that removal of the

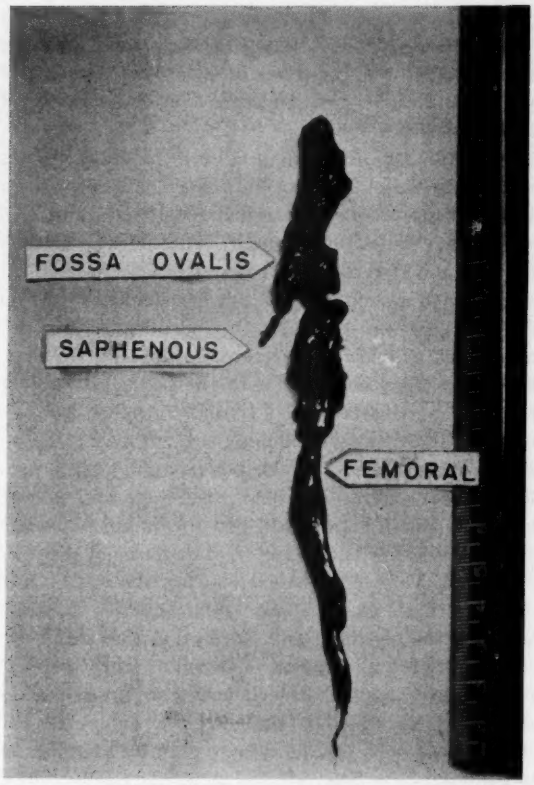

FIG. 4. Thrombus removed from the common femoral, superficial femoral, and external iliac veins in case 1. It is 18 cm. long and it was lifted out without difficulty. The patient recovered with no post-thrombophlebitic edema.

thrombus under these circumstances with closure of the vein would be the better choice of treatment. This offers not only protection against major embolic phenomena but it also reduces the post-thrombophlebitic edema with its resulting disability which unquestionably follows ligation.

CASE REPORTS

Case 1. A 55 year old man, a carpenter by trade, felt a sudden severe pain in his left leg as he was walking. This was followed very promptly by massive swelling of the leg. Pain has been present but it has not been excruciating. Treatment by his referring doctor prior to admission had been rest and elevation of the extremity. Seventy-two hours after onset of symptoms he was admitted to the Baptist Hospital at 6:00 p.m. on July 10, 1952.

There was considerable induration of the tissues, particularly in the thigh and the swelling extended down to and involved the foot. No swelling was found in the abdominal wall. Tenderness along the course of the femoral vein extended up to the groin. His blood pressure was 120/60. The pulse rate was 100 and respiratory rate 20. Routine laboratory work showed normal findings except an elevated leukocyte count (16,150).

On the morning following admission, an operation was done under spinal anesthesia. An incision was made over Scarpa's triangle and the femoral vein was exposed. The common and superficial femoral veins were firm and contained a clot. Small Penrose drains were placed beneath the vein both at the upper and lower ends of the exposed segment. With these any bleeding could be controlled. A longitudinal incision then was made in the vein and a long clot was removed by forcefully exerting pressure along the course of the vein from below upward. A clot 10 cm. long was expressed from the lower segment. Free bleeding occurred. The Penrose drain was tightened to control this bleeding. The vein then was milked down by pressure on the lower abdomen and down towards the thigh. There was another large clot 7 cm. in length expressed from the vein followed by a gush of blood. A small clot 2 cm. long was expressed from the upper end of the long saphenous vein. Theoretically the clot should be expressed down from the upper segment first, then while the vein is controlled by tightening the thin rubber Penrose drain the clot from the lower segment should be expressed. Such a principle is now adhered to. The vein was not ligated, but using no. 00000 silk sutures on atraumatic needles the opening was closed approximating intima to intima by a Carrel stitch. The thigh wound then was closed.

The saphenous vein was isolated just as it crossed the ankle and the upper end of it was intubated with a polythene tube. This was connected with an infusion set containing 5 per cent dextrose. In the first 500 cc. of the infusion only 75 mg. of heparin were added. This was done in order not to change the coagulation time too promptly and to give the operative site a chance to seal and thus avoid hemorrhage. Every hour for a period of 10 minutes the nurse tightened a soft rubber tourniquet on the thigh just above the knee just sufficiently to constrict the superficial venous circulation.

The coagulation time in the next 72 hours varied between 6 and 15.5 minutes and after 72 hours the infusion was discontinued. Approximately 150 mg. of heparin a day were used in the infusions.

In the first 24 hours there was marked improvement. Edema had subsided at least 50 per cent after 24 hours. By the third day the leg looked entirely normal and the polythene tube was removed. Penicillin was given each day (400,000 units).

The patient left the hospital on the fourth postoperative day, July 11, appearing well.

The measurements of the leg were as follows:

	Calf	Thigh above Knee
Circumference		
July 11 right	12 inches	19 inches
left	13½ inches	20 inches
July 14 left	11¾ inches	18¾ inches

Subsequent reports from the home doctor state that this patient is entirely well and has absolutely no residual edema.

Case 2. A 54 year old pipe-fitter's helper was admitted to the Baptist Hospital on July 15, 1952 and dismissed on July 30, 1952. Twenty-five days prior to admission he fell from a height of some 15 feet landing upright on his feet. Able orthopedic surgeons treated him for a fracture of the left os calcis. A cast was applied, extending from his toes to the upper thigh. The day prior to his admission to the hospital, he had sudden swelling of the left lower extremity with sharp pains in the leg. The cast was removed by his orthopedic surgeons.

Examination showed a strongly built man. The temperature was 101.8 F. The left leg was tremendously swollen and edematous. It was a reddish purple color and was hot. The swelling extended from the upper thigh down to and included the foot. The dorsalis pedis

pulse was palpable. The leukocyte count was 13,450 per cu. mm. Otherwise the laboratory work was normal.

We first saw the patient 48 hours after his admission to the hospital. This was 27 days after the accident and 3 days after the onset of the symptoms of thrombosis. On that day, an operation was done. Under spinal anesthesia an incision was made over the fossa ovalis and the femoral vein was exposed. The vein was opened above the junction of the profunda femoris and a clot was found to be present in this segment. The clot could not be removed en masse and it was removed in pieces by exploring forceps and by a catheter introduced into the vein distally and proximally with suction attached to the catheter. It was estimated that most of the clot was removed for a distance of at least 7 or 8 cm. proximal and distal to the incision. After removing the clot, the blood flow was almost normal from below and less than normal from above, but there was some free flow from each direction.

The longitudinal opening in the vein was closed with a running no. 00000 silk with a Carrel type stitch. The vein was not ligated. After closure of the wound, the internal saphenous vein was cannulated at the ankle, using polythene tubing. A continuous infusion was started containing heparin. It was permitted to run at about 20 drops a minute and approximately 150 mg. of heparin were given intravenously daily which maintained an elevated coagulation time. At one time, on July 20, the coagulation time reached 55 minutes, a dangerous and undesirable level. On July 17 it was 9 minutes, on July 18 14.5 minutes, and on July 19 3.5 minutes. On July 21 the heparin and infusion were stopped and the polythene tube was removed.

The postoperative course of this patient was quite dramatic. The edema of the leg and thigh subsided remarkably in the first 48 hours. After the infusion was discontinued he became ambulatory, using compression bandages and gradually increased the time he remained up. He continued to rest in the middle of the day; otherwise was active. The measurements of the circumference of his legs were as follows:

	Midcalf	Ankle
October 13, 1952		
Right	13½ inches	8½ inches
Left	14 inches	8¾ inches

He was permitted to return to his work as a pipefitter's helper on Nov. 3, 1952, and has continued to work without complaint. When examined on April 27, 1953, a note was made that he looks and feels well. There was little edema on the left. The circumference of the right ankle measured 8⅜ inches, and the left 8⅝ inches.

In such an instance as this it is well for the patient to protect himself with a midday rest if it is at all feasible. The rest should be recumbency and with the leg slightly elevated. Twenty to 30 minutes may make a tremendous difference and avoid the accrual of edema and eventual induration.

Case 3. This 45 yeas old restauranteur was in an automobile accident on Sept. 1, 1952. He sustained multiple contusions and brush burns of the right foot, thigh and sacrum and laceration of the skin over the right medial malleolus. He remained one week in a hospital elsewhere. Three weeks later he developed severe swelling of the right leg with pain and a feeling of intense fullness in the leg. He was treated by procaine sympathetic nerve blocks and with anticoagulants. The swelling did not subside. Pain was severe and his leg felt as though it would burst. Four months after the accident, on December 30, he came under our care, and was admitted to the Baptist Hospital.

The right leg and thigh was the site of massive edema. The extremity was so tense and white and swollen from the groin down to and including the foot that it was difficult for him to move about. The appearance was typical of phlegmasia alba dolens. The knee would not flex because of the massive edema. The leg was tender and sensitive.

Considerable vasospasm was associated with the venous obstruction of the right lower extremity and for that reason it was considered that not only was it justified to explore the vein, but that lumbar sympathectomy may help in this particular case even though, as a general rule, it is not beneficial and is not indicated for post-thrombophlebitic edema.

For the operation the patient was placed on his left side. The abdomen and entire right leg was prepared and draped in the field. A transverse incision was made just above the umbilicus and a lumbar sympathetic ganglionectomy was done, removing the second, third, and fourth ganglia. The position was shifted slightly without removing the drapes, turning the patient more into recumbency. A long incision was made over the medial border of the sartorius muscle. The deep fascia was incised and Hunter's canal was exposed. The femoral vein and artery were identified. There was marked inflammatory reaction around the artery and vein. The vein was opened in three different places. The femoral just below the profunda had a white adherent clot obstructing the lumen. Proximal to this there was a soft, red propagating thrombus which was not firmly attached, and distal to the white organized thrombus was another area of soft red thrombus. A catheter with suction attached was inserted and the vein was sucked clear of the propagating thrombus for a distance of several centimeters proximal and distal to the opening. An increased flow of blood was obtained from the vein but it was not regarded as absolutely normal. A new lumen was made in the vein at this level by inserting an obturator through and through the white thrombus. The vein wall was closed using a Carrel type stitch with no. 00000 arterial silk. The vein was not ligated. The wound was closed leaving a drain under the fascia.

Considerable subsequent serosanguinous drainage occurred from this area and it was believed that the fasciotomy might have had some beneficial effect. This patient received penicillin postoperatively (400,000 units daily) but heparin was not used. It was believed that it was a late type thrombus and actually we were surprised to find a soft red thrombus still existent four months after the original onset of thrombosis.

For several weeks following the operation this patient had a diffuse aching pain in the right thigh and leg. He complained bitterly of it. It was regarded as a postsympathectomy traumatic neuritis. It ultimately disappeared completely. Otherwise he had marked and dramatic improvement following the operation. The swelling disappeared almost completely and his leg looked normal at the time of his dismissal on Jan. 12, 1952. Subsequent follow-up (the last June 3, 1953) has substantiated our early appraisal of a striking but admittedly unexpected successful result. He is working. He has no pain and very little edema. No compression bandage or elastic stocking is necessary.

The result was remarkable and satisfactory both subjectively and objectively. He was advised to continue a midday rest in the recumbent position for 30 minutes in order to prevent the daily accrual of edema and the possibility of an indurated leg.

Case 4. A 35 year old woman, an American by birth but who has been living in Peru, came under our professional care on Feb. 23, 1953. On May 1, 1952, she had, when four months pregnant, a sudden mild swelling of the left leg. On July 1, 1952, when six months pregnant, she miscarried. The swelling of the left leg has continued since. There is no pain.

General examination: She weighed 160 pounds and was approximately 5 feet 9 inches tall. The positive features in the examination concerned the left leg which was moderately swollen from the groin down to and including the foot. There was some pitting edema. There was very little induration and the arterial circulation to the feet was normal.

In view of the remarkable result obtained from the patient in Case 3 four months after onset of his thrombosis, the uncertainty of the result was explained and operation was suggested. The patient was admitted to the Baptist Hospital.

On March 2, 1953, an operation was done. The entire left leg, and the left side of the abdomen, was prepared in the field. A lumbar sympathectomy was done. An incision then was made over the medial border of the sartorius muscle and the femoral vein was ex-

posed at the lower end of Scarpa's triangle. In this area the wall of the vein seemed to be thick and the vein was relatively small. The vein was surrounded by adhesions. These adhesions were separated for a distance of 6 or 8 cm. The vein was opened and no free flow of blood was obtained. A small catheter was passed distally and blood could be obtained with the catheter. The catheter was then passed proximally for 4 cm. and met an obstruction. A rigid obturator was then placed in the vein and the obstruction was forced. However, at this time no free flow of blood was obtained. The wound opening in the vein was closed with small interrupted sutures, everting the edges so that the lumen was restored. The vein was not ligated. An incision was then made in the left inguinal region and the common femoral vein was exposed. It was found to be of normal texture and consistency. The vein was separated from surrounding structures for a distance of 4 cm. but it was not opened.

The patient was dismissed from the hospital on March 10, 1953, after an uneventful convalescence. The leg continued to swell a little postoperatively, as much as it had preoperatively. We were not pleased with the immediate result. She was advised to elevate the leg at intervals for several weeks during the mid-morning and mid-afternoon, and thereafter during the middle of the day. She then returned to Lima, Peru, and has not been under our observation, but we have had communications from her, from which we infer she has not been benefited by the surgery. She wrote on March 29, "My leg swells more than it did before surgery. I get uncomfortable. The entire leg swells but the region above the knee becomes quite large."

The failure to obtain improvement in this case was disappointing although not entirely unexpected. There is of course a limit to the time when such active intervention will help. The result in this patient also reaffirms the impotence of sympathectomy by itself to produce improvement in post-thrombophlebitic edema.

Case 5. A white woman aged 48 was working in her kitchen placing supplies on high shelves. The following day there was pain in the right forearm which radiated up into the shoulder region. Shortly therafter her arm and hand became swollen and the extremity felt cold. She consulted a doctor who advised rest. On Feb. 17, 1953, three days after the onset, she came under our care. She was admitted to Touro Infirmary.

The physical examination was essentially negative, with the exception of the following findings. The right arm, forearm and hand were moderately swollen. The right hand was colder than the left hand and a distended axillary vein could be felt in the axilla. The superficial veins over the shoulder were very prominent and distended.

Several hours after admission to the hospital the patient was taken to the operating room and the right axillary vein was exposed. There was much distention of the vein and a longitudinal incision into it was followed by a profuse flow of blood under pressure. This was controlled by rubber tissue previously placed under the vein for this purpose. A clot was removed from the axillary vein and the subclavian vein. This was accomplished by inserting a catheter (no. 10F) proximally into the vein and exerting pressure with an attached suction machine. The incision in the vein was closed with a Carrel type stitch.

An infusion of 5 per cent dextrose containing 200 mg. of heparin in 500 cc. was started in a vein in the opposite arm. Other infusions supplemented this and in 48 hours 550 mg. of heparin were administered. The maximum coagulation time was 11 minutes. Penicillin was also given, 400,000 units daily.

Following the operation, the distention of the veins over the shoulder rapidly diminished, and the edema disappeared from the hand within 36 hours. The patient was discharged on the fourth postoperative day with no edema and slight distention of the veins of the arm. There was, however, some swelling in the axilla due to a hematoma in the wound. The immediate and late results in this case were excellent. At last examination (May 15, 1953) the arm appeared normal and showed no swelling.

The usual course of effort thrombosis is slow and discouraging with persistent edema gradually subsiding over a long period of time. The rapid improvement in this case left

no doubt that the operation had helped to reduce the residual disability and shorten the course of post-thrombophlebitic edema.

Case 6. A 65 year old sales representative, who weighed 230 pounds, developed severe pain in the right upper abdomen which radiated to his shoulder. Roentgenograms showed gallstones. He was admitted to the Baptist Hospital by his family physician on Oct. 12, 1953, two days after onset, and on October 15 we were asked to see him.

At operation on October 15, diffuse peritonitis and an acutely inflamed perforated gallbladder containing stones were found. The gallbladder was removed from the fundus down carefully to identify the cystic and common ducts. Retention sutures as well as routine layer closure were employed.

His immediate postoperative course was satisfactory. He was out of bed on the fourth postoperative day. On the eighth postoperative day he complained of pain in the calf of the left leg and there was slight tenderness, but no definite Homan's sign was elicited. On this meager evidence depo-heparin 300 mg. were administered intramuscularly; and the same dose was repeated on the following day and an additional dose of 200 mg. on the next day. In spite of this the maximum coagulation time was 6 minutes and 20 seconds. He was ambulatory. On October 28, 13 days after operation he was permitted to go home. Two days later his left leg was badly swollen and he was readmitted to the hospital on October 30. On the following morning he complained of a little chest pain but no further evidence to suggest embolism.

At operation on October 31, an incision was made over the lower aspect of Scarpa's triangle. There was considerable edema in the tissues. The superficial femoral vein and artery were separated. The superficial femoral vein was decidedly distended. Two small Penrose drains were placed beneath it and a longitudinal incision was made in it. Bleeding was profuse, but it was all from the lower segment. A catheter with suction attached was inserted into the inner side and the profunda femoris vein was exposed. It, too, was distended and looked no different from the initial appearance of the superficial femoral vein, but a small aspirating needle inserted into it disclosed that it contained a clot. Thus, it appears, it is sometimes difficult to distinguish a thrombosed vein from one which is ectatic from an obstruction above. The opening in the superficial femoral vein was closed with no. 00000 silk using a Carrel stitch. Rubber tissue drains were placed under the deep vein and a longitudinal incision was made in it. A dark red serpentine tenacious clot began to curl out. The leg was milked from the groin down. About 4 cm. of a clot was expressed from above and the vein began to bleed from that segment. Penrose drains were tightened to prevent further hemorrhage. The thigh was then compressed from the knee upward. A very long clot came out of the vein. Some of it was destroyed in removal, but a length of 12 cm. was preserved intact. The opening in the vein was closed.

After closing the thigh wound, an incision was made over the internal malleolus of the same leg and a polythene tube was tied in the saphenous vein. An infusion of 5 per cent dextrose was started and was permitted to run at 10 drops a minute. Heparin, 100 mg., was added to this infusion. Within the next 72 hours infusions containing a total of 700 mg. of heparin were run continuously. The thigh wound oozed slightly and subsequently developed a hematoma, which had a restraining effect on the quantity of heparin given. The maximum coagulation time was 11 minutes and 30 seconds.

Edema of the leg gradually subsided. Examination on Jan. 4, 1954, showed much improvement but some persisting edema. Progressive improvement has permitted his return to work on Jan. 11, 1954.

It would obviously be very interesting and almost a conclusive proof of benefit in such cases if a venogram would show that the deep vein is open and functioning. Whereas this would be a support of the thesis that this is a helpful form of management it would be of no real benefit to the patient and it would carry a

danger in that it too might initiate a new thrombus. For that reason, so far, we have declined to use it.

SUMMARY

A new type of management for deep vein thrombosis is described. It consists of removing the thrombus and closing the vein without ligating it. Heparin in a continuous infusion is administered in a superficial vein distal to the level of thrombectomy. Six cases are reported in which this method was employed. In those in which the thrombus was recent, brilliant results were obtained with very little morbidity and post-thrombophlebitic edema.

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CONSTRUCTION OF VENOUS VALVES*

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The usual surgical approach to therapy of incompetent valves of the veins of the lower extremity has been that of ligation, excision, stripping, or injection, all of which are locally destructive.[1, 2, 4] If valves could be fashioned from the vessels very early before secondary changes occurred in the walls, then plastic reconstruction might be substituted, at least in part, for present methods. With this in mind, a simple method for constructing venous valves has been tried in dogs and in 1 patient.

METHODS

A simple means of forming an intussusception of vein wall was devised which consists of placing mattress sutures of no. 00000 silk on opposite sides of the wall. Two methods of making valves are possible depending on whether each mattress suture is placed in the wall longitudinally (fig. 1A) or transversely (fig. 2A). If placed longitudinally, the leading edge of the intussuscepted portion is anchored at two points when the valves are completed (fig. 1D), whereas the intussusceptum is free at its leading edge with use of the other type of suture. Because it was thought that an incompetent valve might occur with the latter type of procedure, the former operation was done, using the inferior vena cava of 21 dogs.

Intravenous sodium pentobarbital was used for anesthesia, and the abdomen was entered through an incision in the right lower quadrant. The first dog was operated upon Jan. 1, 1952 and the last on June 27, 1952. Autopsies were done at intervals of 1 month to 1.5 years afterward and the veins were carefully examined.

RESULTS

In three of the dogs tricuspid valves were made, but in the remainder bicuspid valves were made and found to be more satisfactory. At the time of operation, there was every indication that the valves functioned. However, in the autopsy material either the valves were very shallow—9 cases—or they had disappeared (fig. 3). Only one, in a dog autopsied 112 days after operation, was considered a reasonably effective structure. The depth of the sinus between valve cusp and vessel wall bore no relation to the time it had been present. One reason for this outcome was undoubtedly the tension required in doing the operation on the inferior vena cava of the dog. In two vessels early in the series the sutures pulled out due to insufficient amount of the vessel wall being included. Thereafter this

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* Aided by a grant from the National Heart Institute, Department of Health, Welfare, and Education.

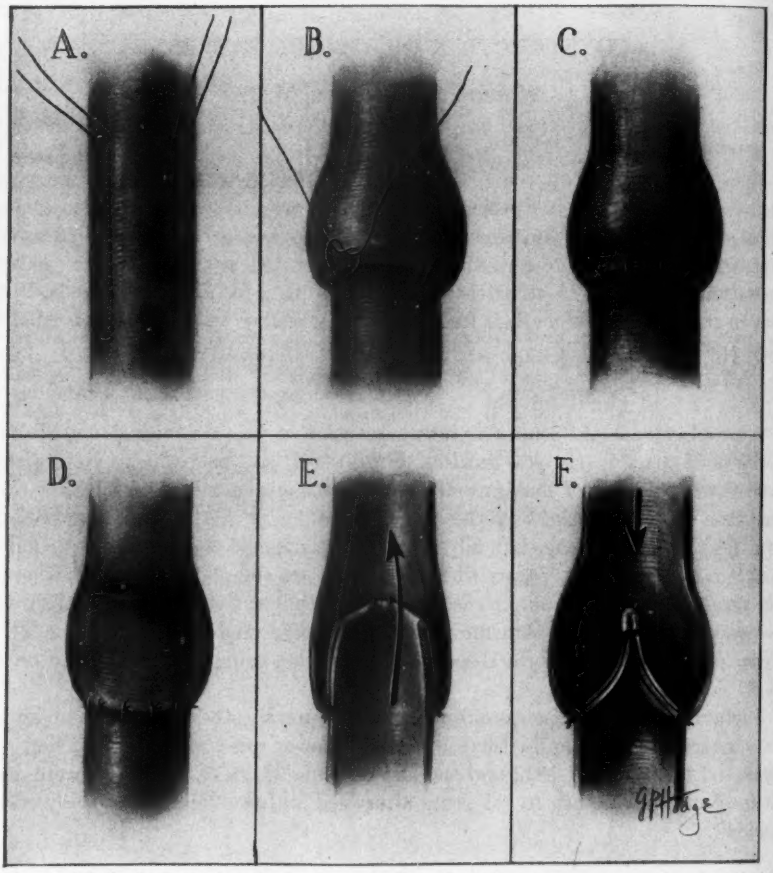

FIG. 1. Method of constructing valves. A. Insertion of mattress sutures longitudinally on opposite sides of the vessel. The distance between points of entrance and exit of the sutures should be approximately 2.5 times the vessel diameter. B. Both mattress sutures have been drawn taut simultaneously and tied. Two additional mattress sutures have been placed in the vessel, bringing the midpoint of the bulge—created by the first two sutures—in the vessel wall down to the adjacent vessel wall. C. The sinus of the valve is completed by tacking its inferior portion to the circumference of the vessel. D. Transparency showing the appearance of the completed valve formed by intussusception of vessel wall. E. Section through the valve (at right angle to figure D) showing it open. F. Section through the valve showing how reversal of flow should cause the valve to close.

fault was corrected. There were no antemortem thrombi in any vessel, and microscopic sections showed an intact endothelial lining (fig. 4). The silk in the vessel wall had elicited only a minimal reaction in sections of vessel wall.

CASE REPORT

The operation was performed clinically for the first time on May 19, 1952. The patient was a 60 year old, white, male laundry employee who had been seen regularly in the Vascu-

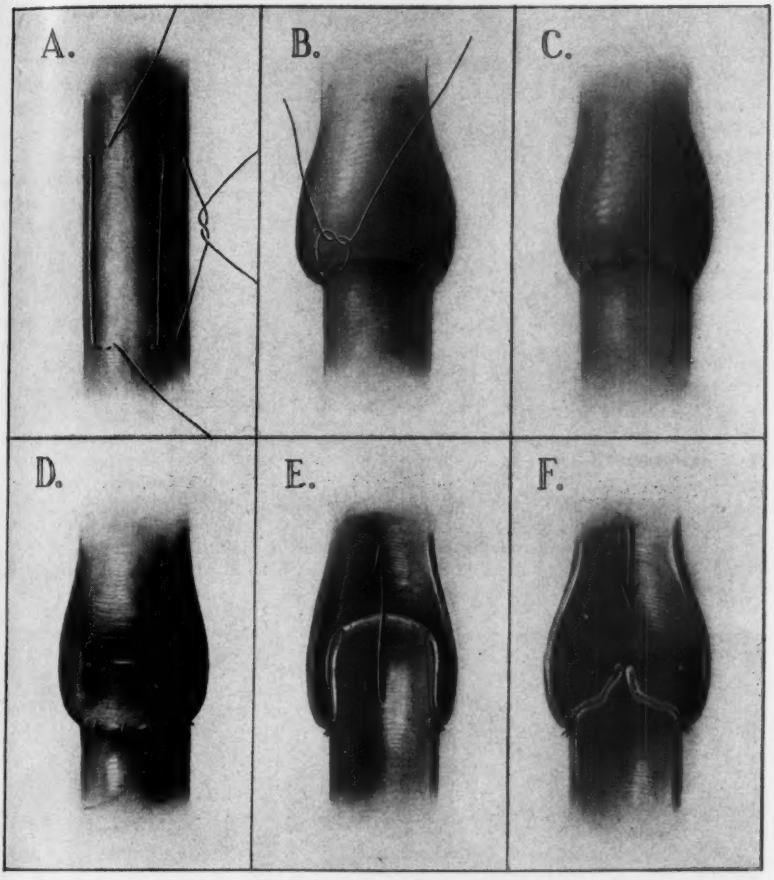

FIG. 2. Alternate method of constructing valves. A. Mattress sutures placed transversely in wall on opposite sides of vessel. B. Mattress sutures pulled taut and tied simultaneously, invaginating a portion of vessel wall approximately 2.5 times its diameter. C. Completion of circumferential row of sutures. D. Transparency showing intussusception of wall with free leading edge. E. Section showing valve open. F. Section showing theoretical closure of valve with retrograde flow of blood.

lar Clinic of Charity Hospital since 1949. He said he had been troubled with varicose veins for at least 11 years and 10 years previously had had a varicose ulcer of the right leg.

He first came to the medical clinic with complaints of weakness, fatigue, anorexia, weight loss of 20 lbs. in 2 years, and malaise. Tuberculosis was suspected, but no active disease was found after careful studies in the hospital during August 1949. He was referred to the vascular clinic because of varicose veins with an ulcer over the right ankle. Thirty-two admissions to this clinic are recorded from this time to May 1952. Treatment consisted chiefly of cleansing the ulcer by various means, a nonadherent dressing, ace bandage, and occasionally a trial with an Unna paste boot. The treatment he received reads as an interesting study of the character of the various physicians who attended him, but the ulcer

FIG. 3. Gross appearance of valve in dog several months after operation. Sinuses (arrows) are present beneath the leaflets but are rather shallow.

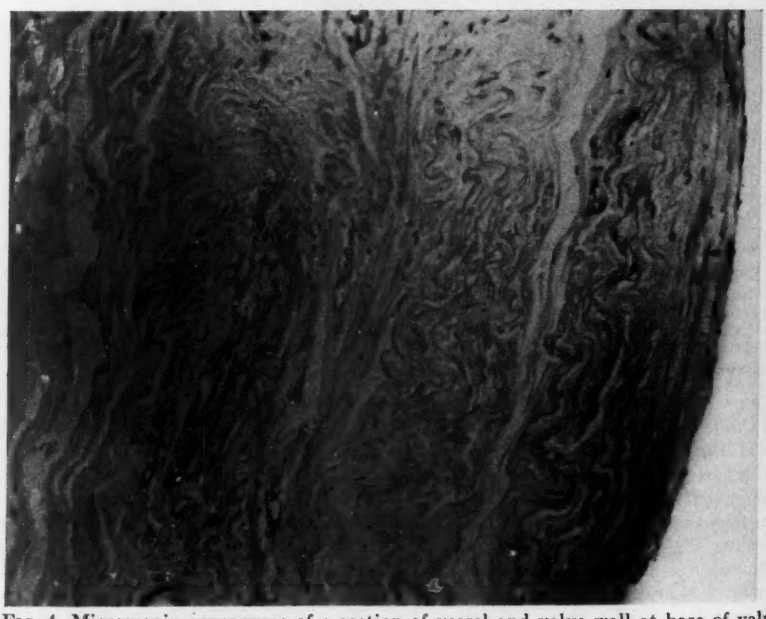

FIG. 4. Microscopic appearance of a section of vessel and valve wall at base of valve leaflet. Note intact endothelium.

did not heal under any regimen. Surgery was refused by the patient on two occasions during this period.

Physical Examination: The blood pressure was 125/90, pulse 64, respirations 18, and temperature 98.6 F. The patient was very frail, thin, and apprehensive. Other than scattered crepitant râles audible over both lung fields the chief findings on physical examination were confined to the right leg. A chronic, indurated, shallow ulcer was present over the right ankle with brown discoloration of the surrounding skin, and numerous varicosities were prominent over the leg, knee, and lower thigh. The long saphenous vein was large, somewhat tortuous, and contained no competent valves. Incompetent perforating vessles were demonstrable at several levels in the lower thigh and upper leg and edema was present, but was not pronounced. No ulceration and only a few varices were present on the opposite side.

Laboratory Findings: The results of blood and urine examinations were within normal limits. Kline: Negative. Roentgenograms showed emphysema, fibrotic infiltration of both lung apices, uncoiling of aorta, and cardiac enlargement compatible with left ventricular hypertrophy. Fluoroscopy: No evidence of an aortic aneurysm. Electrocardiogram: T_1 low. T_v 3, 4, 5 inverted. Q_v 2, 3, 4, 5 present. QRS interval 0.09 sec. There was definite evidence of myocardial disease compatible with anterolateral myocardial infarction, healed.

Operation: Under local, procaine anesthesia valves were fashioned in the main trunk of the long saphenous vein in the upper, middle, and lower thigh, using the technic illustrated in figure 1. Superficial tributaries were ligated at the upper termination of the vessel.

FIG. 5. Appearance of leg after construction of three valves in long saphenous vein in the thigh and multiple low ligations. Note site of healed ulcer.

The vessel was found to have a much thicker wall than indicated on physical examination and this hampered the procedure. A perforating vessel was ligated near the inferior valve. Special care was taken to have the line of approximation of valve leaflets parallel to adjacent fascial planes in order to facilitate function of the structure during exercise.

Course: The wounds healed satisfactorily, but due to incompetent perforating vessels in the leg it was necessary to do low ligations on three occasions subsequently. The ulcer slowly but steadily improved and healed completely, edema disappeared, and the leg appeared as shown in figure 5 when the patient was examined 14 months after the first operation. Examination of the thigh indicated that the saphenous vein was patent and no incompetence was detected. Varices communicating with the short saphenous vein were seen, but further surgery was not advised in view of the patient's cardiac status.

DISCUSSION

The valves which at first seemed satisfactory in the dog failed to persist as functioning structures. The tension necessary for making the valves and the horizontal position of the vena cava in a quadruped are factors which may have contributed to the outcome. Although these results were disappointing, some information was gained from the study. A bicuspid valve was found more satisfactory technically than a tricuspid valve. It was easy to visualize and guide the needle properly in constructing the valve, and little trouble was experienced from bleeding even though the suture passed entirely through the wall of the vessel. It was found best to draw up the paired sutures on either side of the vessel simultaneously, so tension could be maintained while each suture was tied. Endothelium promptly covered the sutures, and no difficulty from thrombosis was experienced. The thin-walled vessels in the dog were much more suitable for the procedure than the saphenous vein with thickened wall encountered in the clinical case. The tissue included in the mattress sutures must be generous to prevent them from pulling out, but of course undue tension on the suture line is as undesirable in this as in any other surgical procedure.

The case study suggests that it may be possible to construct satisfactory valves in patients, but the report of 1 case in the presence of abundant clinical material indicates the conservative viewpoint taken. Preservation of the superfical system might be possible rather than the commonly accepted practices of high ligation and stripping if further studies demonstrate that such venous valves will function. This is particularly true in view of the infrequent occurrence of incompetent communicating vessels in the thigh.[3] The greater saphenous vein in man is a most suitable vessel for trial in well selected cases; smaller veins, deep veins, and communicating vessels offering more obstacles to success. Perhaps at best such an operation can be only a part of the entire surgical therapy of the patient with incompetent venous circulation, but a successful procedure for constructing valves in veins and arteries is a goal which, if achieved, would have wider implications than solution of problems associated with varicose veins.

SUMMARY

A method for constructing venous valves was devised, but tests in 21 dogs showed that the valves in the inferior vena cava persisted as an adequate structure in only 1 instance.

Valves were constructed in the long saphenous vein and multiple low ligations were done in 1 patient with varicose veins. No evidence of incompetence of the vessel was found after a period of 14 months had elapsed.

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THE MANAGEMENT OF CLINICALLY OBSCURE INTRATHORACIC LESIONS

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The last decade has provided the chest physician with an imposing array of new diagnostic aids together with increasing refinement of older procedures. Standard roentgenographic technics, body section roentgenograms, bronchoscopy, bronchography, angiocardiography, sputum cytology, bacteriologic methods and biopsies of easily accessible tissues, have all found their places in the diagnostic approach to diseases of the chest and their value has been demonstrated beyond question.

However, in spite of the employment of any or all of these procedures, many intrathoracic lesions remain indeterminate. The management of these clinically obscure lesions recently has become a topic of considerable discussion among students of pulmonary disease and there is a growing conviction that a specific diagnosis must be quickly established in such cases, if all other diagnostic methods have failed, this, of course, requires that an exploratory thoracotomy be done with direct biopsy of the lesion. In order to determine the wisdom of this seemingly radical approach to the problem, it is necessary to establish several facts: (1) the urgency of early diagnosis in such cases; (2) the accuracy of diagnostic impressions based on complete clinical evaluation compared to the accuracy of diagnosis resulting from exploratory thoracotomy; and (3) the hazards of the operative procedure. In an effort to further clarify this situation, the authors have analyzed their own experiences with such lesions.

During the 7½ year period from May 1946 to November 1953, 226 thoracotomies—exclusive of procedures for empyema drainage, reexpansion of pneumothorax, and transthoracic approach for subdiaphragmatic disease—were done on the Thoracic Surgery Service of the Veterans Administration Hospital, New Orleans, Louisiana. Of the number, 100 thoracotomies (44.2 per cent) were done in cases in which the exact nature of the pulmonary or mediastinal lesion could not be determined by the usually employed diagnostic methods. All of the patients were males whose ages ranged from 19 to 70 years, with the great majority falling within the fourth to the sixth decades.[1] An analysis of these 100 cases disclosed that malignant neoplasms were present in 50 per cent; 45 per cent of the patients had bronchogenic carcinoma; 2 per cent had primary malignant tumors of other types, and 3 per cent had metastatic lesions from extrathoracic primary sites (table I).

Bernatz and Clagett[2] encountered bronchogenic carcinoma in 30 per cent of 180 cases. Grow and associates,[5] in a series of 200 cases composed chiefly of younger individuals, reported malignant tumors in 21.5 per cent of their cases.

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TABLE I

Exploratory thoracotomy

Type Lesion	*No. Cases*
Bronchogenic carcinoma	45
Lung abscess	14
Tuberculoma	7
Pulmonary tuberculosis	6
Bronchiectasis	3
Lipoid granuloma	2
Blastomycosis	2
Sarcoid disease	2
Chronic pneumonitis	2
Coccidiodomycosis	2
Echinococcus cyst	1
Pulmonary infarct	1
Chronic interstitial fibrosis	1
Malignant thymoma	1
Malignant metastatic melanoma	1
Metastatic choriocarcinoma	1
Bronchial adenoma	1
Lymphosarcoma	1
Aortic aneurysm	1
Pleural hibernoma	1
Cystic disease of lung	1
Pericardial cyst	1
Metastatic adenocarcinoma	1
Pleural mesothelioma (benign)	1
Teratoma	1
Total	100

The incidence of primary bronchogenic carcinoma in this series is even higher, and is considered to represent ample evidence that a correct early diagnosis is urgently needed in such cases, since it is well established that the patient's chances of survival are directly related to the rapidity of diagnosis and immediate institution of resective therapy. This aspect of the situation alone seems to justify the employment of exploratory thoracotomy in almost all cases in which the character of a pulmonary lesion cannot be established by more conservative methods. The value of early thoracotomy is further attested by the fact that 24 (53.3 per cent) of the 45 bronchogenic carcinomas encountered were resectable (table II). This figure represents a significant increase in resectability over the

TABLE II

Bronchogenic carcinoma

	No. Cases	Percentage
Resectable	24	53.3
Nonresectable	21	46.7
Total	45	100.0

TABLE III

Pulmonary resections

Type	Malignant	Benign	Total
Pneumonectomy	22	9	31
Lobectomy	4	20	24
Segmental—wedge	1	15	16
Exploration—without resection	23	6	29
Total	50	50	100

report of Ochsner and associates, in which it was found that resection was possible in only 35 per cent of 948 cases.[7]

The remaining 50 per cent of the cases in this series was composed of benign tumors or inflammatory lesions comprising 18 separate pathologic conditions (table I). In a significant number of these cases the disease state found on exploration was of such a nature that resection represented the therapy of choice. This is clearly brought out by the fact that the exploratory procedure was followed by pulmonary resection or local removal of the lesion in 44 (88 per cent) of 50 cases (table III) and is substantiated by the report of Brewer and coworkers,[3] who analyzed 30 cases with benign lesions in which the diagnosis was established at exploratory thoracotomy. They concluded that in 23 of the 30 cases cure could not have been expected by conservative means. Twenty-seven were cured by resection of the lesion either by lobectomy or local excision.

When supporting diagnostic aids fail to provide the clinician with a specific answer in dealing with an intrathoracic lesion, his clinical impressions frequently will be erroneous in a significant number of cases. In this series the preoperative diagnosis, based on all evidence at hand, was not corroborated by the final histologic diagnosis in 46 per cent of the cases. This fact is of definite significance, in that all cases were submitted to a joint medical-surgical chest conference and the presurgical impression thus represented the combined opinions of a number of specialists in chest diseases.

It becomes obvious then, that clinical diagnosis cannot be relied upon if it is to be in error in almost one-half of the cases. This situation results, in part, from the fact that standard diagnostic studies, while usually helpful, are at times misleading. In some instances, bronchoscopic examination, laminograms, and bronchograms were suggestive of bronchial neoplasm when a malignant process was *not* present and the lesion proved to be inflammatory in nature.

On other occasions, acid-fast organisms were found in the secretions of the respiratory tract or in gastric washings, and seemed to point to a diagnosis of tuberculosis, when in fact these organisms were actually unrelated to the true underlying process. In this series, acid-fast organisms were found in a variety of patients in which the primary pathologic condition included a pericardial cyst, blastomycosis,[4] bronchogenic carcinoma, and nonspecific pneumonitis. The true identity of these organisms frequently was never established. In other cases, they were found to be nonpathogenic acid-fast bacilli or acid-fast fungi.

It is emphasized that in many cases isolated findings must be interpreted in the light of the entire clinical picture before their significance can be determined. If one incautiously accepts the demonstration of acid-fast organisms as evidence of tuberculosis in all cases, many other disease states, including bronchogenic neoplasm, will be overlooked. A high index of suspicion must be maintained, particularly when roentgenographic findings are somewhat atypical.[8] Frequently, the final answer can be obtained only by resort to exploratory thoracotomy. The coexistence of tuberculosis and bronchogenic carcinoma is another hazard to correct diagnosis, and the physician must be aware that the presence of one by no means rules out the other.

The roentgenographic demonstration of calcium within an otherwise indeterminate pulmonary lesion is another finding which may lead the clinician to an erroneous conclusion. It is frequently stated that the presence of calcification is situations of this kind is indicative of benign disease.[6] While this may be true in most instances, there are some exceptions to this concept. The authors have seen 2 patients—not included in this series—in which calcified nodes or areas of intrapulmonary calcification, resulting from previous inflammatory disease, had become incorporated within the substance of a bronchogenic carcinoma. If these lesions had been considered to be benign on the basis of this finding and exploratory thoracotomy deferred, the diagnosis certainly would have remained in error until an inoperable state ensued. It is further emphasized that even when the presence of calcium is assumed to indicate a benign process, but does not disclose the exact nature of the lesion, thoracotomy often will be indicated. The authors accept only one type of calcification which they consider to be indicative of a specific benign pulmonary entity. This is the typical concentric laminated target-like calcification which is found in some tuberculomas. The tendency to rely solely on the roentgenographic appearance of pulmonary

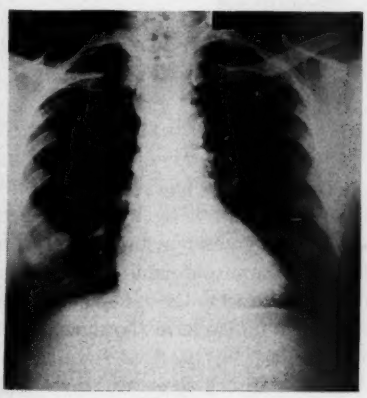

FIG. 1

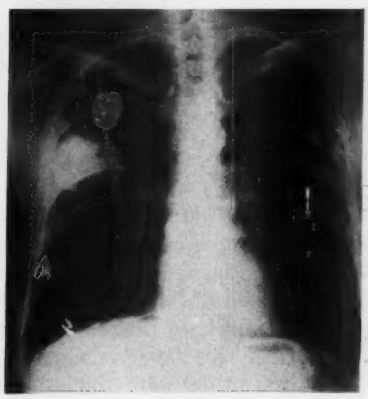

FIG. 2

FIG. 1. A blastomycotic granuloma of the right lower lobe simulating a peripheral bronchogenic carcinoma.

FIG. 2. A pulmonary infarct, which has assumed a nodular configuration, and could not be differentiated from a neoplasm.

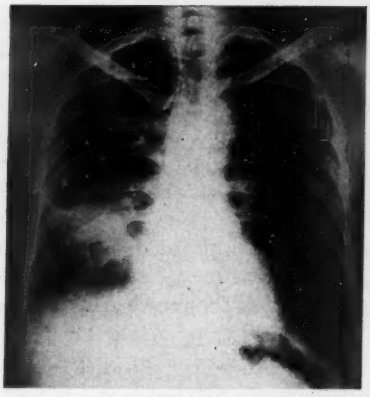

FIG. 3

FIG. 4

FIG. 3. Lipoid granuloma of right middle lobe with complete bronchial occlusion, simulating bronchogenic carcinoma.

FIG. 4. Lipoid granuloma of right middle lobe with complete bronchial occlusion, demonstrated by bronchogram.

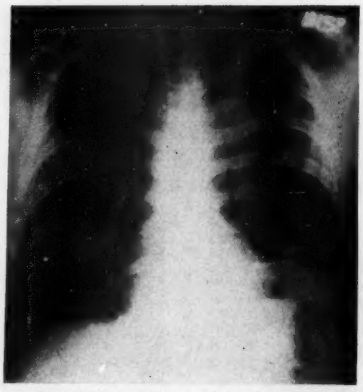

FIG. 5. Bronchogenic carcinoma in a 29 year old white man

lesions as a definite indication of malignancy or benignancy is not supported by the evidence at hand (figs. 1 to 5). Many physicians are content to let time settle their diagnostic dilemma, but we are firmly convinced that slow diagnostic methods or prolonged periods of observation have no place in the management of the patient with indeterminate lesions of the lung.

In sharp contrast to the failure of less direct methods to establish a specific diagnosis is the outstanding accuracy of exploratory thoracotomy. In this series, an exact diagnosis was determined by the operative procedure in 95 per cent of the cases. In 3 instances the surgeon was so confident that an inoperable

neoplasm was present that he elected not to remove tissue for biopsy. In the 2 remaining cases, biopsy of mediastinal lymph nodes proved to be negative. It is obvious that a diagnosis might have been established in all cases if tissue for biopsies had been taken and if frozen sections had been requested.

Biopsy of either the involved segment or the lobe itself should be the procedure in a lesion that does not have mediastinal involvement. If mediastinal nodes are present, they should be biopsied primarily. If these prove negative for malignancy, as shown in some of our cases, the involved pulmonary segment or lobe should be excised for histologic examination.

The hazard of exploratory thoracotomy may best be illustrated by a study of operative mortality. In this series, 7 deaths were considered due to operations. However, this figure undoubtedly misrepresents the true situation, since in 4 of these cases, thoracotomy was accompanied by pneumonectomy and the mortality rate is believed to be more directly related to the resective procedure. The remaining 3 deaths occurred in elderly, poor-risk patients, all ofm who were found to have extensive inoperable bronchogenic carcinomas. In no case did an operative death occur in a patient with nonmalignant disease who had exploration alone. The authors are of the opinion that the hazards of exploratory thoracotomy detract little from the value of the procedure, and in no way prohibit its use. We are led to agree with the statement of Bernatz and Clagett[2] who, after reporting an operative mortality of 1.7 per cent, concluded that: "The risk of exploratory thoracotomy today certainly is not more than that of exploratory laparotomy, and no longer is the policy of *wait and see* justifiable. The physician, not time, should make the diagnosis of pulmonary disease."

SUMMARY AND CONCLUSIONS

One hundred cases are reviewed in which exploratory thoracotomy was done after extensive diagnostic studies had failed to establish the exact nature of an intrathoracic lesion.

An unusually high incidence (50 per cent) of malignant tumors was encountered, and primary bronchogenic carcinoma was present in 45 per cent of the cases.

When inflammatory disease was found, the lesion usually was a type best treated by surgical excision. The exploratory procedure was accompanied by resection in 88 per cent of these cases.

Preoperative diagnostic impressions based on all evidence at hand frequently were in error (46 per cent).

Exploratory thoracotomy established a specific diagnosis in 95 per cent of the cases. This figure might have been brought to 100 per cent in biopsy of suitable material had been taken in every case.

An operative mortality of 7 per cent in this series is believed to be more directly related to accompanying major resective surgery, and should not exceed 3 per cent in thoracic exploration per se.

The authors have concluded that procrastination usually is unwise in dealing with indeterminate pulmonary lesions; that an early correct diagnosis is impera-

tive, and that exploratory thoracotomy should be employed almost without exception in such cases.

The authors wish to express their appreciation to Mr. Harris Prentis for his kind assistance in compiling statistical data used in this paper.

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DESMOID TUMORS

A REPORT OF ELEVEN CASES

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The term *desmoid tumor* first appeared in the literature in 1838 when Müeller[21] introduced it to designate tumors with a tendon-like consistency. Today *desmoid tumor* refers to a well established clinical and anatomic entity which is defined as a benign fibrous tissue neoplasm that originates in the fascia or aponeurosis about striated muscle, and which has the peculiar characteristic of locally invading and engulfing adjacent muscle tissue. Below is presented a brief historical survey and a summary of current concepts of this interesting but comparatively rare group of neoplasms. We add to the cases of *desmoid* tumors previously reported, 11 cases from Charity Hospital of Louisiana at New Orleans.

Macfarlane[19] is given credit for having first described *desmoid* tumors. However, 1 of the 2 cases he reported in 1832 was labelled "organized sarcomatous tumor between the layers of the abdominal muscles" and the other was referred to as "fibrocartilaginous tumor" of the abdominal wall.

Although Müeller introduced the term *desmoid* tumor in 1838, this name was not applied specifically to the neoplasms under discussion until 1880–1884 when Sänger[29] published a series of articles in which he called these growths *desmoid* tumors. Early the fibrous and benign nature of this tumor was recognized and prior to Sänger's publications the terms *fibroids*, *fibroma* and *fibroplastic tumor* were used to designate these neoplasms. German, English and American authors followed Sänger in the use of the term *desmoid tumor* but French authors persisted in calling these tumors *fibromas of the abdominal wall*. In one respect the French authors were more correct, as *desmoid* has sometimes been mistakenly used to include definitely malignant tumors.

For many years it was the general opinion that *desmoid* tumors occur only in the abdominal wall. It is now accepted that *desmoids* are not peculiar to the abdominal wall, but that they occur in other striated muscles. This was first recognized and reported by Guyon[13] in 1877 but little attention was given this fact until 1923 when Nichols[25] reported six extra-abdominal *desmoid* tumors with histologic proof of their similarity to abdominal *desmoids*.

Not a small part of the discussion of *desmoids* has been concerned with the point of origin of the tumor. Muscle tissue, periosteum, uterine round ligament, the aponeurosis and sheath of muscles and the intermuscular septa all have been implicated. Huguier[15] in 1860 advocated the periosteal origin of these tumors and was supported in his theory by Bodin[2] in 1861 and Nélaton[23] in 1862. The musculoaponeurotic theory of origin was advocated by Cornils[7] in 1865, Buntzen[6] in 1868 and Boye[4] in 1873. Ebner[9] in 1880 and Hertzog[14] in 1883 stated the

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origin to be in ruptured muscles. Labbe and Remy[16] in 1888 suggested that muscular rupture during violent labor contractions was important and compared the tumor to callus and keloid. Guinard[12] in 1910 suggested the origin to be from the uterine round ligament. Wyllys Andrews[1] in 1911 called attention to the development of these tumors in postoperative scars. Sänger[30] suggested in 1884 that *desmoids* arise from the aponeurotic sheath of the abdominal muscles and not from muscle tissue directly. At present, these neoplasms are considered benign fibrous tissue tumors or fibromas that have their origin in the fascia or aponeurosis about muscle or from the fibrous tissue between the muscle bundles.

INCIDENCE

Desmoid tumors are comparatively rare. Their infrequency is attested by reports from foreign literature. Guerlt[11] found only 8 cases in 16,000 tumors reviewed from the files of Viennese hospitals. Pfeiffer[28] recorded only 40 cases at von Brun's Clinic in Tubingen in 46 years. Labbe and Remy[16] observed but 10 cases in 20 years.

The English literature is comparable in regard to the incidence of these tumors. Stone[32] reported 5 cases from 21,000 surgical patients treated at Johns Hopkins. From the Mayo Clinic over a period of 40 years 130 cases are reported by Musgrove and McDonald.[22] Pack and Ehrlich[25] found 17 cases among 50,346 cases of neoplastic disease admitted to Memorial Hospital between the years 1917 and 1943. In the same report Pack and Ehrlich state that, in their survey of all tumors in the anterior abdominal wall, *desmoids* account for 3.5 per cent of all tumefactions, 4.2 per cent of all true neoplasms, 5.5 per cent of all primary tumors and 7 per cent of all benign tumors.

SEX AND AGE

Approximately 80 per cent of these tumors occur in females. *Desmoid* tumors occur at any age, but most commonly occur in the third and fourth decades. Approximately 10 cases of congenital *desmoid* tumors have been reported in the literature. The earliest recorded tumor of this type was noted at birth and reported by Brockman.[5]

ETIOLOGY

The etiology of *desmoid* tumors is not known. The majority occur in the abdominal wall of women who have borne children and attempts have been made to relate the tumor to pregnancy. The relative rarity of this tumor compared with the frequency of pregnancy suggests that there is no direct relationship.

One of the most attractive theories offered is the traumatic theory. Three varieties of trauma are considered of etiologic importance: (1) physiologic trauma of the abdominal wall caused by pregnancy, (2) direct trauma, and (3) trauma secondary to abdominal operations.

The findings of Geschickter and Lewis[10] and of Lipschutz and his co-workers[18] suggest the possibility of an endocrine origin of *desmoid* tumors. Geschickter and

Lewis, in a study of the hormone content of various connective tissue tumors, found a fairly high concentration of gonadotropic substances in *desmoid* tumors. Lipschutz and his co-workers have produced fibromyomas in the abdominal wall of guinea pigs with estrogenic substances. These fibromyomas behave like *desmoid* tumors in that they invade and destroy adjacent muscle tissue, but do not metastasize. The experimentally produced tumors regress when estrogen administration is stopped and antifibromatogenic hormones (testosterone, progesterone and desoxycorticosterone) can prevent their formation.

SYMPTOMS

Desmoid tumors as a rule are asymptomatic. The majority are discovered accidentally; frequently a minor trauma calls the patient's attention to the mass. After discovery of the tumor subjective symptoms frequently develop. These neoplasms most commonly develop in otherwise healthy young women who have borne one or more children. *Desmoids* vary in size from 8 to 20 cm. in diameter as an average, and their growth is slow and gradual. Tumors of large size may cause pressure symptoms.

Abdominal *desmoids* present the findings of an intramural tumor. The skin is not fixed to the mass. With the abdominal muscles relaxed, there is enough mobility of the tumor to indicate nonfixation to the intraperitoneal organs. With deep breathing the mass moves forward but not up and down, as do intraperitoneal tumors. Contraction of the abdominal muscles fixes the mass—Bouchacourt's sign.[3] With intraperitoneal tumors, contraction of the abdominal muscles causes the mass to become less prominent whereas with masses in the skin and subcutaneous tissues, fixation of the muscles causes an increase in prominence of the swelling.

Approximately 75 per cent of reported *desmoids* are in the abdominal wall,[22] the remainder are found in various muscles and muscle groups about the body. The more common extra-abdominal sites are the pectoralis major muscle, rectus femoris muscle, gluteal muscles and muscles about the scapula. Both abdominal and extra-abdominal *desmoids* occur in postoperative scars (Penick[27]).

GROSS CHARACTERISTICS

Desmoid tumors usually are solitary and unilateral. As the name implies, the tumor is a dense, hard, fibrous growth. It cuts with difficulty, imparting a grating sensation to the knife. The cut surface bulges, is glistening white or pinkish in color and presents interlacing whorls or bundles of white fibrous tissue, an appearance not unlike that of uterine leiomyomas. The center of the tumor, where the blood supply is the poorest, may show softening.

The growths may appear to be circumscribed or they may be diffusely infiltrating. All show infiltration characterized by interdigitation of tumor with skeletal muscle. The circumscribed growth usually is round or oval, with the long axis of the mass parallel to the long axis of the muscle involved.

A true capsule is absent, but, due to pressure on the surrounding structures, a pseudocapsule may be seen. *Desmoids* may become adherent to the peritoneum

or to the periosteum but they do not invade these structures. They do not extend through the subcutaneous tissue to involve the skin.

MICROSCOPIC FINDINGS

The microscopic picture of *desmoid* tumors is that of a nonencapsulated fairly cellular fibroma that has infiltrated striated muscle and engulfed striated muscle fibers. The tumor is somewhat more cellular at its periphery where it infiltrates adjacent muscle tissue. Lymphocytic infiltration is seen in this region. Blood vessels are chiefly the adult type and are less abundant at the center, where myxomatous or cystic degeneration is frequently noted.

The special feature of *desmoids* that differentiates these tumors from simple fibromas is the inclusion of striped muscle fibers in various stages of atrophy. An excellent study of the changes in the engulfed muscle fibers is presented by Stewart and Mouat.[31] They divide these changes into three phases: 1. Early changes noted at the periphery of the tumor where engulfed muscle fibers become stretched, irregular in outline and begin to lose their transverse striations. The nuclei remain in their normal peripheral location. 2. An intermediate stage where muscle fibers become elongated, thin, shrunken with nuclei evenly distributed throughout the cytoplasm. They resemble at this stage unstriped muscle fibers. 3. In this stage the cytoplasm swells; becomes homogeneous and strongly eosinophilic with enlarged rounded or oval nuclei peripherally distributed. They resemble very closely tuberculous or foreign body giant cells. Stewart and Mouat expressed the belief that these changes are due to faults of nutrition and innervation and represent a vegetative or nonfunctioning existence on the part of the muscle fibers or fragments. They refer to these changes as a de-differentiation or reversionary change similar to the plasmoidal regression of striped muscle described by Durante[8] in 1902.

Although these tumors show infiltration into adjacent muscle tissue, there have been no reports of lymph node involvement or of distant metastases and *desmoids* are properly considered benign tumors. In some reports reference is made to sarcomatous changes in these neoplasms. According to authors who have recently presented exhaustive treatises on this subject, (Musgrove and McDonald,[22] Stewart and Mouat,[31] Perlman and Mayo,[26] Moore and King[20]), there is no evidence that *desmoids* undergo sarcomatous changes.

DIFFERENTIAL DIAGNOSIS

In the majority of cases, the problem of diagnosis is not a particularly difficult one. In very stout or muscular individuals and in instances where the tumor is deeply located, the differential diagnosis may be most difficult and frequently biopsy is necessary to determine the nature of the tumor. In the differential diagnosis of *desmoid* tumors a number of conditions must be considered and for purpose of discussion they may be grouped as follows. 1. Tumors of the skin and subcutaneous tissues. 2. Tumors of muscle and muscle sheath. 3. Tumors of periosteum and of bone. 4. Intraperitoneal tumors.

Tumors of the skin and subcutaneous tissue include dermatofibromas, keloids,

neurofibromas and lipomas. These are superficially located tumors and do not become fixed on contraction of underlying muscle. Many are adherent to the skin and some are multiple.

Tumors of muscle and muscle sheath include fibrosarcomas, hematomas, diffuse subacute myositis, hemangiomas and rhabdomyomas.

Fibrosarcomas present a difficult problem in differential diagnosis. They are more frequently encapsulated, larger in size and softer in consistency than *desmoids*. Microscopically, they present a higher degree of cellularity; more mitotic figures and a greater variation in size, shape and staining of nuclei. Numerous pathologic mitoses and tumor giant cells are seen which are not noted in the *desmoid* tumor. Metastases from fibrosarcoma, locally to lymph nodes, distally to lung, liver and bone are not unusual.

Hematoma usually has a history of sudden onset following trauma; early it is fluctuant; later it is crepitant and when organized it becomes firm. Roentgenographic studies may reveal calcification in an old hematoma.

Diffuse subacute myositis may give a clinical picture similar to *desmoid* tumors and somewhat similar pathologic findings. It can be distinguished by the diffuse infiltration of inflammatory cells as compared to the limited peripheral distribution of lymphocytes seen in the *desmoid* tumor.

Hemangiomas and rhabdomyomas are rarer entities and the differential diagnosis depends upon microscopic findings.

In the differentiation of *desmoid* tumors from tumors of periosteum and bone, roentgenographic studies are helpful. When a *desmoid* tumor is attached to the periosteum the differential diagnosis may be particularly difficult.

Intraperitoneal tumors are less prominent when the abdominal muscles contract. On deep respiration, intraperitoneal tumors as a rule move up and down. *Desmoids* do not move up and down, but seem to move forward.

TREATMENT

The treatment of *desmoid* tumors resolves itself into three different approaches: 1. Surgery, which is the principal method of treatment. 2. Irradiation, which is auxiliary or palliative. 3. Hormone therapy, which is essentially experimental.

Surgery: In consideration of the surgical treatment of these tumors, it must be remembered that, although *desmoid* tumors are benign, they do invade adjacent muscles and may become attached to periosteum and to peritoneum. If tumor tissue is left behind a local recurrence of the neoplasm develops. The surgeon, therefore, should not be satisfied with mere enucleation of a *desmoid* tumor, but should make an excision that is wide and well beyond the boundaries of the tumor. This may require resection of a large part of the abdominal wall, parietal peritoneum, Pourpart's ligament or periosteum. In extra-abdominal *desmoids*, the tumor may be adjacent to or surround major nerves and blood vessels. This may influence the surgeon to do a less radical operation than he should and lead to a high recurrence rate.

Early excision of the tumor is advocated. If excision is done while the tumor is small, it is possible to avoid a problem of difficult closure of the wound. The

methods of repair of defects that follow radical excision of these tumors depend on the problem at hand. Simple approximation of the fascia, overlapping of the fascia as in Mayo's repair of umbilical hernia, the use of a fascial or muscular pedicle graft or a free fascial graft have all been employed. Tantalum mesh particularly is useful in the repair of large defects.

Irradiation: There is little evidence to show that irradiation has any beneficial effect other than to hold the tumor in check. Pack and Ehrlich[25] suggested that the beneficial results of radiation therapy may be due to incidental castration brought about during treatment of lower abdominal wall lesions. Radiation therapy should never be used when the lesion can be extirpated surgically. It should be reserved for the poor-risk patients, for patients in whom the lesion cannot be completely removed and for patients who develop recurrence after surgical excision.

Hormone Therapy: The findings of Lipschutz and his co-workers[18] suggest that hormone therapy should have a place in the management of these tumors. We are unable to find a report in which the antifibromatogenic hormones were used in the management of *desmoid* tumors.

PROGNOSIS

Desmoid tumors are benign. They have the property of locally invading adjacent muscle, but they do not metastasize and they do not undergo malignant degeneration. A cure can be effected by complete excision; however, if excision is inadequate, the tumor recurs locally. Complete excision of the recurrent tumor will effect a cure.

The recurrence rate after removal of abdominal *desmoids* was quite high in early reports, 30 to 40 per cent. In a more recent report from the Mayo Clinic,[26] a 6 per cent recurrence rate is reported. The lower recurrence rate is due to a better understanding of the nature of the tumor and the more radical excision now employed.

Recurrence after removal of extra-abdominal *desmoid* tumors is much more frequent than after removal of the abdominal variety. Musgrove and McDonald[22] in 1948 report a 77 per cent recurrence rate in their follow-up of 26 patients with extra-abdominal *desmoid* tumors. The probable cause of the high recurrence rate is commented upon earlier in this paper.

REPORT OF CASES FROM THE CHARITY HOSPITAL OF LOUISIANA AT NEW ORLEANS

Eleven patients with *desmoid* tumors were operated upon at Charity Hospital in New Orleans between the years 1940 and 1953. Nine of these had *desmoid* tumors of the abdominal wall. Table I records the salient features of these cases. Among these, Case M. T. was operated upon by one of the authors, and figure 1 is a photograph of the gross specimen removed from this patient; figure 2 is a photomicrograph of the same specimen.

Two of the 11 patients had extra-abdominal *desmoid* tumors. Both of these had recurrence of their tumor after excision, and in both cases the recurrent

TABLE I

Summary of nine cases of abdominal desmoid tumors. Charity Hospital of Louisiana at New Orleans, 1940–1953.

Case, Year	Sex, Age	Duration before Operation	Size of Tumor	Number of Pregnancies	Muscle Involved	Location	Follow-Up	Remarks
F. T. 1940	C., M. 52	4 years	21 by 15 by 9 cm.	—	Rectus abdominis	Right upper abdomen	1 year	No recurrence
H. G. 1940	W., F. 49	3 years	11 by 9 by 6 cm.	8	Rectus abdominis	Lower abdomen, scar	None	Desmoid reccurred in supra-pubic area
T. S. 1941	W., F. 22	5 years	11 by 10½ by 4½ cm.	3	Ext. int. oblique and rectus	Left lower abdomen	8 years	No recurrence
H. S. 1943	C., F. 39	1 year	6 by 5 by 3 cm.	1	Rectus abdominis	Below and rt. umbil.	None	None
E. B. 1943	W., F. 24	7 mos.	11 by 8 by 7 cm.	0	Sacrospinalis	Right flank	None	Occurred in an incision and drainage scar
I. H. 1944	C, F. 26	6 mos.	8½ by 6 by 5 cm.	2	Int. oblique, rectus abdominis	Left lower abdomen	7 years	No recurrence
J. B. 1944	W., F. 34	2 years	3½ by 3½ by 4½ cm.	0	Ext & int. oblique	Right abdomen	1 year	No recurrence
M. T. 1952	C., F. 23	16 mos.	5½ cm. in diameter	3	Left rect. abdominis	Left abdomen	1 year	No recurrence
E. G. 1951	C., F. 17	1 year	16 by 16 by 10 cm.	0	Rectus abdominis	Lower abdomen	None	None

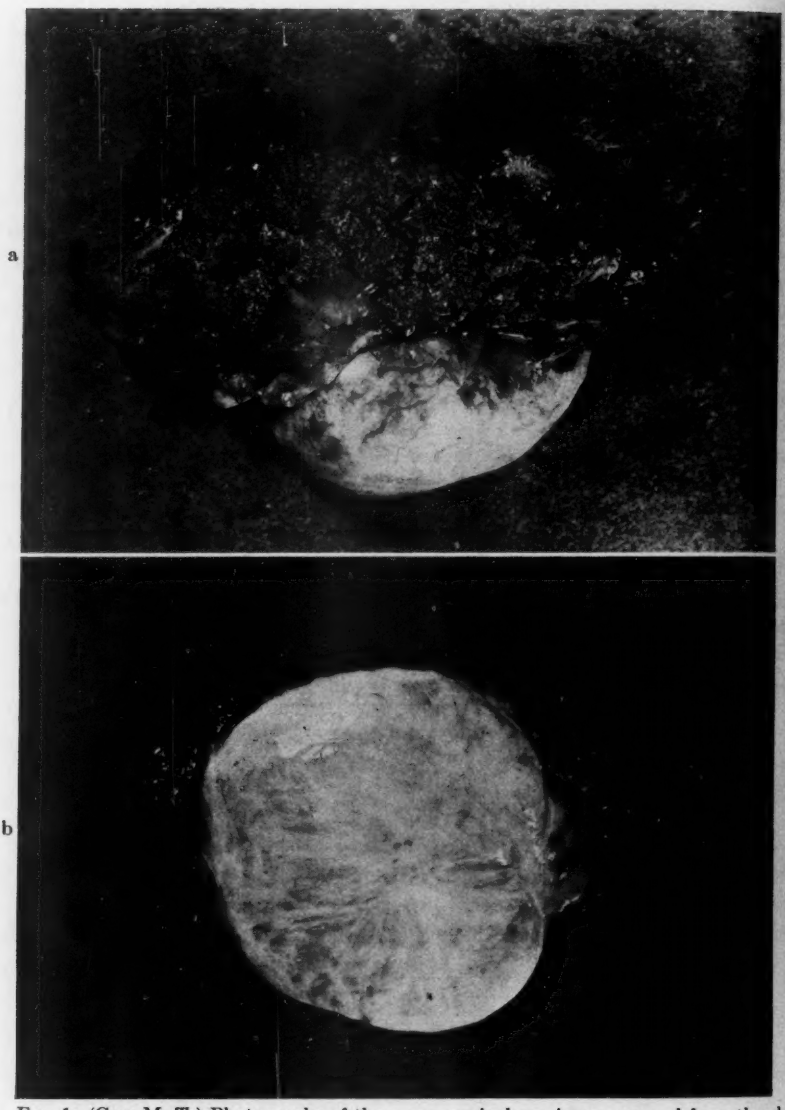

FIG. 1. (Case M. T.) Photographs of the gross surgical specimen removed from the abdominal wall showing (a) desmoid tumor with surrounding muscle and peritoneum, (b) cut section of same tumor, note its similarity to uterine leiomyomata.

tumor was excised. One has been followed 9 months, the other 14 months after the second operation, and so far they are free from a second recurrence. These 2 cases illustrate the difficulty encountered in the management of extra-abdominal *desmoid* tumors commented on earlier in this paper. Extra-abdominal *desmoids*

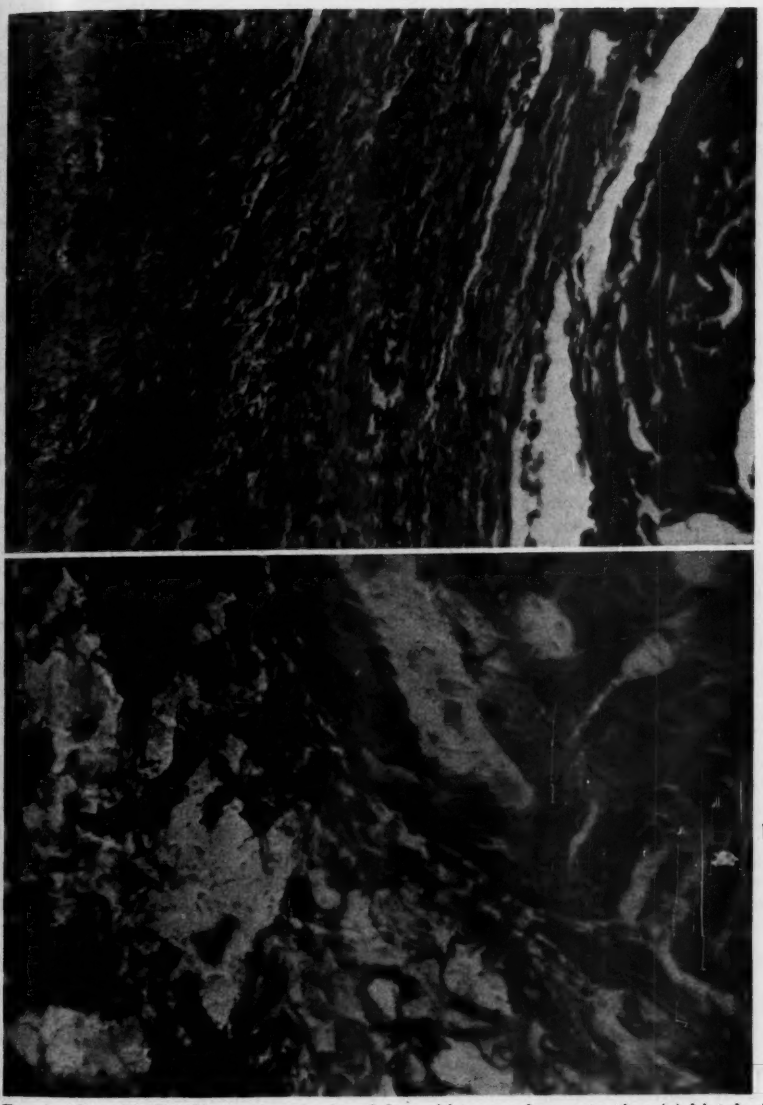

FIG. 2. (Case M. T.) Photomicrographs of desmoid tumor demonstrating (a) histologic structure of neoplasm with adjacent striated muscle, (b) transitional changes in the striated muscle at the periphery of the tumor. Note the multinucleated giant cells.

are likely to surround major nerves and blood vessels. This may lead to a conservative excision with incomplete removal of the tumor. These 2 patients may have been benefited by irradiation after the first excision since in both cases it was thought by the operator that some tumor tissue was left behind. A short summary of these 2 cases follows:

CASE REPORTS

Case 1. S. G., a white woman, 56 years of age, was admitted to Charity Hospital July 19, 1948. She gave a history of a mass in the left arm of six weeks duration. She thought the mass related to injections received in the arm two years before. Examination revealed a hard mass, irregular in outline, located on the lateral surface of the left arm. The mass was not fixed to the skin or bone, and was slightly tender. Moderate swelling of the fingers and hand was noted. An operation was done August 6, and a mass involving the triceps muscle was found. The tumor was attached to the periosteum of the humerus and surrounded the radial nerve. The growth was dissected free from the radial nerve and from the periosteum of the humerus. Part of the triceps muscle was excised. The pathologist reported extensive fibrosis in skeletal muscle with chronic inflammatory reaction. The patient was discharged Aug. 11, 1948

On Jan 15, 1953 this patient was readmitted to the Charity Hospital. According to the history the patient noted a recurrence of the tumor of the left arm in 1950. The mass was becoming gradually larger. On Jan. 17, 1953 the left arm was operated upon for the second time. The mass was found to involve the long and short heads of the triceps and to surround the radial nerve. The long head of the triceps was partially excised, the short head totally excised, and the radial nerve was dissected free from the mass and preserved. The pathologic report on this specimen is *desmoid* tumor, and review of the first specimen reveals findings compatible with a *desmoid* tumor. The patient was discharged February 2. She was last seen in the clinic on Oct. 19, 1953, at which time she was free from recurrence.

Case 2. C. M., a 58 year old Negro woman was admitted to the Charity Hospital at New Orleans on May 3, 1951. She had noted a mass in her neck three months prior to admission. The mass had grown slowly and was located just below the right mastoid process. On May 5, 1951 operation was done. The mass was found to involve the right sternomastoid muscle and was not fixed to the skin or deeper structures. The vagus nerve was dissected free, and left intact. Branches of the spinal accessory nerve involved in the mass were excised and so was a part of the internal jugular vein. In the operative dictation the operator expressed the opinion that some tumor tissue had been left behind. The pathologist reported the specimen to be an extra-abdominal *desmoid*.

In June, 1952, approximately one year after the first operation, the patient noted a recurrence of a mass in the right side of her neck. Growth was slow. She was admitted to the hospital Aug. 5, 1952. Examination revealed a mass located beneath the scar of the previous operation. The mass was firm and slightly movable. A second operation was done on Aug. 15, 1952, which consisted of a radical neck dissection with removal of the entire sternomastoid muscle, the internal jugular vein and the tip of the parotid gland. The pathologist described a 4.5 by 3.5 by 2 cm. mass involving the sternomastoid muscle and made the diagnosis of extra-abdominal *desmoid*. This patient was last seen in the clinic on Oct. 14, 1953, at which time there was no evidence of recurrence.

SUMMARY AND CONCLUSIONS

Desmoid tumor is an established clinical and anatomic entity.

The current concepts are that a *desmoid* tumor is a benign fibrous tissue neoplasm that originates in the fascia or aponeurosis about striated muscle; that it has the peculiar characteristic of locally invading adjacent muscle, but does not metastasize; that it does not undergo sarcomatous changes.

Desmoid tumors occur most commonly in the abdominal wall, but they do develop in extra-abdominal striated muscle.

Complete excision of a *desmoid* tumor effects a cure, but if any tumor tissue remains a local recurrence develops.

Early and radical surgical excision is the treatment of choice. Irradiation is

of questionable value and hormone therapy has not had an adequate clinical trial.

The recurrence rate after removal of extra-abdominal *desmoid* tumors is much higher than after removal of the abdominal variety. Factors that contribute to this higher recurrence rate are discussed.

Eleven cases are reported from the Charity Hospital of Louisiana at New Orleans.

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TRAUMATIC PANCREATITIS*

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Pancreatic injuries of traumatic origin are of three types: operative, penetrating, and nonpenetrating.

The role of surgical trauma in the production of acute pancreatitis has been impressively demonstrated by the recent excellent studies of Warren,[9] Dunphy[4] and others.[2, 3, 6] Operative injuries are responsible for most cases of traumatic pancreatitis.

Penetrating wounds of the pancreas are relatively uncommon in civilian practice.

The importance of nonpenetrating injuries to the abdominal wall as a cause of acute pancreatitis has not received sufficient emphasis. At the Charity Hospital in New Orleans, nonpenetrating or so-called subcutaneous injuries to the pancreas comprise 5 per cent of the cases of acute pancreatitis. A recent review[1] of 109 consecutive cases of acute pancreatitis observed at this institution during the past decade disclosed that in 6 patients the pancreatitis had been initiated by blunt trauma to the abdominal wall. The present report is based on a study of these 6 cases of traumatic subcutaneous injury to the pancreas.

CASE REPORTS

Case 1. M. S., a 36 year old Negro man, was admitted to Charity Hospital two hours after an automobile accident in which his epigastrium had been struck by the steering wheel. Examination revealed dyspnea, hematemesis, epigastric tenderness and rigidity, and signs of peripheral circulatory collapse. A diagnosis of ruptured spleen was made, and, after the administration of 1000 cc. of blood, laparotomy was done. The pancreas was found to be completely divided at its neck, and there was brick bleeding from the laterated pancreas (fig. 1). An estimated 1000 cc. of fresh blood were present in the peritoneal cavity and in the lesser omental sac. There were multiple hematomas in the walls of the stomach and transverse mesocolon. The cut surfaces of the pancreas were closed with mattress sutures of cotton, and a large rubber drain was placed down to the pancreatic defect. After a stormy postoperative course complicated by fever, paralytic ileus, and a copiously draining external pancreatic fistula, the patient recovered and has remained well for three years.

Case 2. E. A., a 42 year old woman, entered the hospital a few minutes after an automobile accident in which she had sustained a steering wheel injury to the epigastrium. She had been unconscious following the accident, but by the time she reached the hospital she presented no signs of serious injury. Although there was slight generalized abdominal tenderness, she did not have abdominal pain, and she was permitted to go home. Twenty-four hours later she suddenly developed severe left upper quadrant pain, epistaxis, hemoptysis, hematemesis and melena. Upon her return to the hospital she was found to have generalized abdominal distention, tenderness and rigidity. The hemogram was normal except leukocytosis was present (17,000 cells). Urinalysis disclosed a 2 plus glycosuria. A diagnosis of ruptured viscus with peritonitis was made. Laparotomy revealed a greatly en-

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Presented before the Southeastern Surgical Congress, Birmingham, Alabama, March 8, 1954.

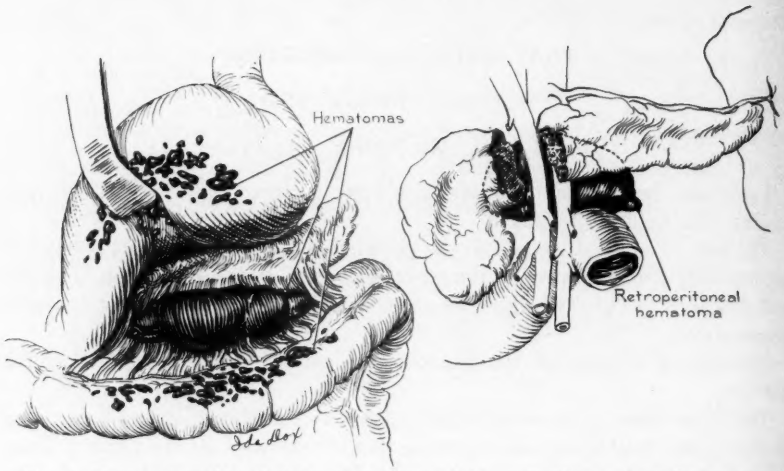

FIG. 1. Case 1: Complete division of pancreas with massive hemorrhage following nonpenetrating injury to the abdomen.

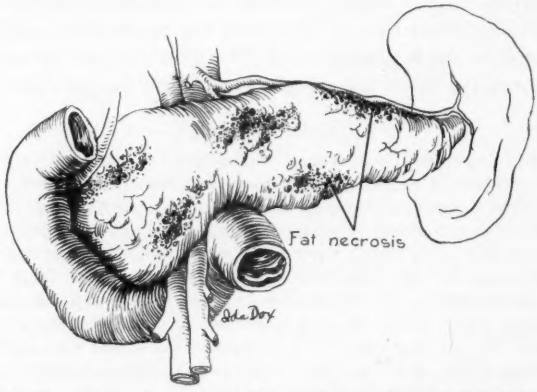

FIG. 2. Case 2: Contusion of pancreas with swelling, edema and fat necrosis involving pancreas and contiguous structures in a patient who had sustained a steering wheel injury to the epigastrium.

larged, edematous, swollen pancreas with fat necrosis involving the pancreas and surrounding structures (fig. 2). Approximately 1 liter of *prune juice* fluid was present in the peritoneal cavity. Unfortunately a serum amylase determination was not made until the fourth postoperative day when it was found to be normal. The patient recovered.

Case 3. A. P., a 16 year old female, was admitted to Charity Hospital 48 hours after an automobile accident in which she was struck in the epigastrium by the steering wheel. Initially she had experienced only mild discomfort in the upper abdomen, but four hours after the accident she became unconscious for a few minutes. When she regained consciousness she complained of progressive increase in the severity of upper abdominal pain and tenderness. Vomiting, abdominal distention and obstipation appeared. When seen in the emergency room 48 hours after the accident, she appeared gravely ill, and there were marked distention, tenderness, and rebound tendernessand spasm in the epigastrium and right flank.

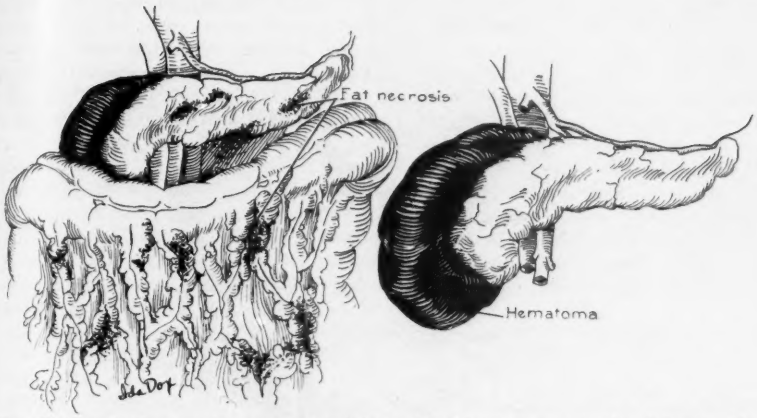

FIG. 3. Case 3: Traumatic pancreatitis initiated by blunt trauma to the abdomen. Note hemorrhagic mass in head of pancreas with fat necrosis involving pancreas, retroperitoneal tissues, omentum and transverse mesocolon. The serum amylase was 473 units.

A scout film of the abdomen revealed a single dilated loop of small bowel containing a fluid level. The hemogram was normal except leukocytosis was present (18,000 cells). The serum amylase was 473 units,* and it remained elevated for six days. The preoperative diagnosis was laceration of the liver with intraperitoneal hemorrhage. Laparotomy revealed approximately 1000 cc. of serosanguinous fluid in the peritoneal cavity, and there was a large firm hemorrhagic mass in the head of the pancreas with fat necrosis involving the pancreas, retroperitoneal tissues, omentum and transverse mesocolon (fig. 3), Multiple rubber drains were placed about the pancreas. Two weeks later it was necessary to incise and drain a large pseudocyst of the pancreas. The resulting pancreatic fistula persisted for three weeks. The patient has remained well.

Case 4. G. F., a 30 year old man was seen in the emergency room 12 hours after he had fallen and struck his abdomen while he was intoxicated. He had experienced immediate onset of severe epigastric pain and tenderness which had progressively increased in severity. Examination revealed generalized abdominal tenderness, spasm and rebound tenderness, these findings being most pronounced in the right upper quadrant. Urinalysis, hemogram and a scout film of the abdomen were negative. The preoperative diagnosis was ruptured intra-abdominal viscus. At operation 500 cc. of bright red blood were found in the retroperitoneal space (fig. 4). There was a deep laceration of the posterosuperior portion of the head of the pancreas. The extravasation of blood had extended into the gastrohepatic and gastrocolic omenta. Drains were appropriately placed about the lacerated pancreas. This man recovered and has remained well.

Case 5. C. W., an 8 year old girl was struck in the epigastrium by a turnstile at an amusement park. She complained of slight abdominal discomfort, but the injury was considered inconsequential. Six hours later, she began to experience severe epigastric pain and tenderness accompanied by nausea and persistent vomiting. Eight hours following the accident she entered the hospital where examination revealed a pale, acutely ill child with generalized abdominal tenderness and rigidity; these signs being most pronounced in the left upper quadrant. A scout film of the abdomen was negative. After the administration of 1500 cc. of whole blood the hematocrit was 48, and white blood cell count was 16,000 per cu. mm. The urinalysis showed a 1 plus albuminuria. A diagnosis of ruptured spleen was made. At laparotomy 500 cc. of fresh and old clotted blood were encountered in the peritoneal cavity

* Normal in this laboratory 80–120 units.

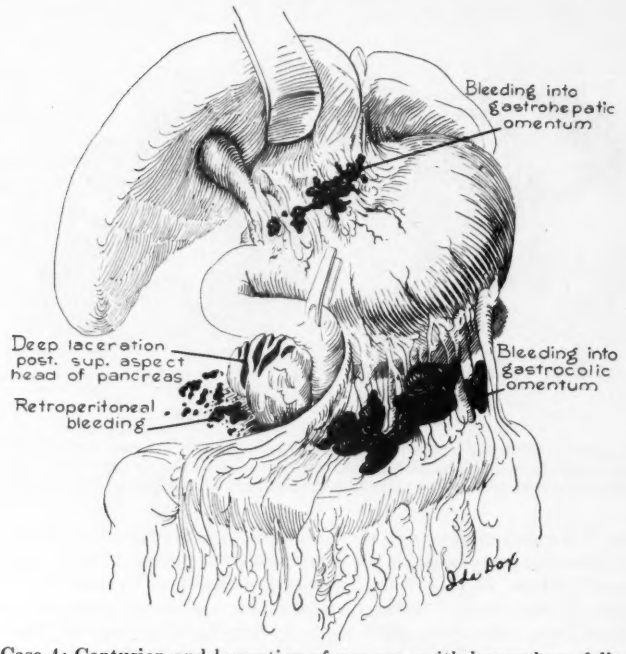

FIG. 4. Case 4: Contusion and laceration of pancreas with hemorrhage following nonpenetrating injury to abdomen.

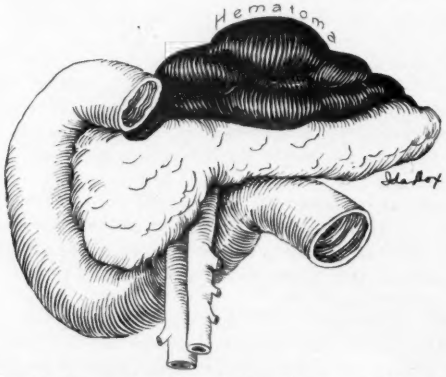

FIG. 5. Case 5: Massive hemorrhage from injured pancreas in a child who was struck in epigastrium by a blunt object. Serum amylase was 417 units.

(fig. 5). There was a huge hematoma occupying the retroperitoneal space over the entire upper half of the abdomen. The superior border of the body of the pancreas was the site of a hematoma, although active bleeding had ceased. After partial evacuation of the pancreatic hematoma several gelfoam sponges were applied to the pancreas and a large Penrose drain placed down adjacent to the injured organ and led out through the gastrocolic omentum and through a stab wound in the left upper quadrant. Another 1500 cc. of blood were

administered while she was in the operating room. Her early postoperative course was smooth except for three or four days of mild ileus. Drainage was slight after the first 48 hours, and on the eighth postoperative day, the drain was removed, and she was permitted to go home. Forty-eight hours later the patient was readmitted to the hospital because of fever of 102 F. The pulse was 114, and the child appeared acutely ill. There was a small quantity of purulent malodorous drainage on the dressing which had been placed over the left upper quadrant stab wound. The hemoglobin was 16 Gm. the white blood cell count 40,000 per cu. mm. and serum amylase 417 units. A rubber catheter was inserted into the stab wound in the left upper quadrant, and by roentgenographic examination the tip of the catheter was found to lie in close proximity to the first lumbar vertebra. Sixty cc. of thick yellow pus were evacuated through the catheter. Treatment consisted of the parenteral administration of antibiotics and daily injection of large quantities of penicillin through the catheter into the retroperitoneal space. The child was discharged 23 days later and has had no further difficulty.

Case 6. M. A., an 8 year old girl, was brought to the hospital a few minutes after being injured in an automobile accident. Examination disclosed unconsciousness, cyanosis, severe dyspnea, blood pressure 50/0, right tension pneumothorax, and a simple fracture of the left mandible. The child became conscious a few minutes after the pneumothorax had been adequately treated and the rapid administration of blood had partially corrected the shock. Although she did not complain of abdominal pain, examination of the abdomen revealed distention with spasm, tenderness, and rebound tenderness which were most pronounced in the epigastrium and right upper quadrant. Paracentesis done in the right flank yielded bright red blood. Twelve hours after admission the serum amylase was 290 units, and 24 hours later it was 214. Two liters of whole blood were administered in a 12 hour period. Because of the multiplicity and gravity of the associated injuries the abdominal injury was treated conservatively. Improvement was rapid except for transient icterus and a troublesome paralytic ileus which responded to gastric suction. She was dismissed from the hospital 15 days later. When last seen several months later she seemed entirely well.

DISCUSSION

There were no deaths in this small series of 6 cases of traumatic pancreatitis, although all of the patients were gravely ill. In each case the pancreatitis had been initiated by blunt trauma to the epigastrium. A prominent feature in one-half of the cases was the delayed appearance of severe symptoms. Massive hemorrhage from the injured pancreas was found to have occurred in 4 of the 5 patients submitted to laparotomy. Multiple serious associated injuries precluded operation in 1 case. Major complications included external pancreatic fistula in 2 cases, a pseudocyst in 1 case and an abscess in or about the pancreas in 1 instance. The serum amylase was elevated in 3 of the 4 cases in which the test was made.

While the pancreas occupies a deep and seemingly protected position in the abdomen with its head and part of its body lying at the level of the first and second lumbar vertebrae, the structure, vascularity and fragility of the organ are such that even minor trauma may result in serious injury. The pancreas may be contused, lacerated or completely divided by a sudden epigastric blunt force which drives the organ against the unyielding bony spine.

Venable[8] suggested a pathologic classification of pancreatic rupture into two types: (1) incomplete rupture in which the laceration involves only the parenchymal portion of the gland without capsular disruption; and (2) complete rupture in which both the capsule and the parenchyma are torn. In either instance the pancreatic ducts may be ruptured or the external pancreatic secre-

tions forced through the duct walls into the interstitial tissues; the pancreas continues its active secretion, and the extravasated pancreatic digestive ferments exert a necrotizing effect upon the blood vessels and parenchyma of the gland.

The patient with an incomplete rupture of the pancreas initially may consider the injury inconsequential, but as extravasated blood and acinar secretions tightly distend the glandular capsule the pain becomes severe hours or days later. If and when the capsule ruptures, the lesser peritoneal cavity suddenly is flooded with highly irritating juices with resultant intensification of signs and symptoms. In the event of complete rupture of the pancreas there may be immediate massive hemorrhage from torn vessels coupled with the early escape of activated ferments into the surrounding tissues, and the patient may be gravely ill from the very beginning. These wide variations in the pathologic process partially explain the lack of uniformity in the clinical picture presented by patients with traumatic pancreatitis.

The potential complications of pancreatic injuries include: (1) acute pancreatic edema and pancreatic necrosis; (2) immediate or delayed massive hemorrhage; (3) pancreatic cysts; (4) pancreatic abscess; (5) internal and external fistulas; (6) diabetes mellitus; (7) hypocalcemic tetany; (8) chronic relapsing pancreatitis and (9) pancreatolithiasis.

There are no classical signs or symptoms indicative of injury to the pancreas. In many cases of traumatic pancreatitis there are multiple injuries to adjacent upper abdominal viscera, and the signs from trauma to these other structures may overshadow those arising from the pancreas. It is suggested, however that the diagnosis can be made in most instances if the surgeon will remain aware of the possibility of pancreatic injury in all cases of trauma to the upper abdomen, and will obtain repeated serum amylase determinations under such circumstances.

Even if it were possible to make a clinical diagnosis of traumatic pancreatitis on the basis of the clinical findings and an elevated serum amylase value, usually operation still would be indicated because of the probability of associated injuries to other abdominal viscera. This is not to deny that operation is unnecessary in most cases of pancreatitis following nonpenetrating abdominal injury if serious injury to other organs and massive hemorrhage and disruption of major pancreatic ducts can be ruled out but it is my belief that usually such cases cannot be predicted with sufficient accuracy to justify a conservative nonoperative approach.

The operative treatment of traumatic pancreatitis will in most instances be limited to the control of hemorrhage; the evacuation of hematomas and adequate drainage of the lesser sac. The exact procedure employed will, of course, depend upon many factors such as the type and extent of the injury, and the condition of the patient. In most instances of incomplete rupture of the pancreas, simple drainage of the lesser omental bursa will suffice. If complete rupture exists the laceration should be closed by nonabsorbable sutures, if this is feasible. When the tail of the gland is hopelessly injured it is better to excise it, and close the stump of the body of the pancreas with interrupted mattress sutures. If the gland is completely torn in half each end should be carefully closed with mat-

tress sutures after which the gap which remains may be loosely plugged with gauze or merely with a large Penrose tube or sump drain. The recognition and implantation of the divided duct of Wirsung into a segment of jejunum or an anastomosis between the ends of the severed duct rarely will be feasible at the time of the emergency operation.

The proper postoperative management of these patients is of the greatest importance. This involves the aggressive application of the same therapeutic principles which are followed in the conservative nonoperative management of any patient with severe acute pancreatitis. These include: (1) the relief of pain; (2) correction of fluid, electrolyte and blood volume deficits; (3) prevention of infection; (4) relief of abdominal distention; (5) reduction in the volume of pancreatic secretions; (6) detection and treatment of disturbances in glucose metabolism; (7) prevention of hypocalcemia and (8) detection and ultimate surgical elimination of pancreatic cysts and abscesses.

The pain of acute pancreatitis may be excruciating and unresponsive to the usual doses of narcotics. It usually is necessary to employ these narcotics despite their objectionable tendency to induce spasm in the sphincter of Oddi. Banthine seems to be helpful in relaxing the sphincter of Oddi and diminishing pancreatic secretion.

Dr. Mims Gage has been largely responsible for stimulating the surgical members of the Charity Hospital staff to employ paravertebral block in the management of pancreatic pain, and by this procedure it frequently has been possible dramatically to relieve the pain of acute pancreatitis. Orr and Warren[7] reported the application of continuous epidural procaine block in these patients and consider it a more satisfactory method of pain relief.

Appropriate parenteral fluids, electrolytes and blood must be administered to restore and maintain normal blood volume and electrolyte balance. Dehydration results from the persistent vomiting and the loss of blood; pancreatic secretions and components of the intra-abdominal inflammatory exudate is sometimes tremendous. Recent evidence has been presented to indicate that the intravenous administration of human serum albumin is beneficial in that it contains an antitrypsin factor.[5]

Antibiotics should be administered because of the tendency for abscesses to form in and around the pancreas following traumatic pancreatitis.

Paralytic ileus is a troublesome complication in almost all cases of acute pancreatitis. Its development should be anticipated and prevented by the early insertion of the Miller-Abbott tube into the small intestine.

Starvation, continuous gastric suction, and the administration of banthine should diminish the secretion of pancreatic enzymes.

Diabetes mellitus occasionally accompanies acute pancreatic necrosis, and if unrecognized may be responsible for progressive acidosis and death of the patient following operation.

Large quantities of calcium may be deposited in foci of fat necrosis with the resultant development of hypocalcemia. If this complication is not recognized the occasional patient with massive pancreatic necrosis will due of hypocalcemic tetany. Therefore repeated blood calcium determinations should be carried out,

at least after the third day, and calcium gluconate administered intravenously if calcium deficit is demonstrated.

A pancreatic pseudocyst may appear within a few days following nonpenetrating injuries to the pancreas, or it may be weeks or months before this complication becomes evident. It should be managed by marsupialization or internal drainage, and in rare instances by excision. Small pseudocysts frequently can be cured by simple incision and drainage.

The development of an abscess in or about the pancreas should be suspected in the patient with acute traumatic pancreatitis who, following a period of clinical improvement, develops fever and other signs of infectum.

SUMMARY AND CONCLUSIONS

Six cases of acute pancreatitis resulting from blunt trauma to the abdominal wall are reported. There were no deaths.

Nonpenetrating or so called subcutaneous injuries of the pancreas are responsible for about 5 per cent of cases of acute pancreatitis at the Charity Hospital in New Orleans.

Following trauma to the upper abdomen the possibility of pancreatic injury must always be considered, and serial determinations of the serum amylase should be done in such cases.

It should be possible to make a correct preoperative diagnosis in most cases. Only at operation, however, can associated injuries to other abdominal viscera and the disruption of major pancreatic vessels and ducts be recognized and properly treated.

In operating upon patients in whom one or more of the upper abdominal viscera have sustained injury the surgeon should examine the pancreas before closing the abdominal wound, otherwise injuries to this organ frequently may be overlooked.

While this series is much too small to justify any definite conclusions, it does seem possible that cases of acute traumatic pancreatitis form a special group in which the natural history of the disease may not be entirely comparable to that of the usual form of acute pancreatitis.

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A PRELIMINARY STUDY OF THE NUMBER AND DISTRIBUTION OF VALVES IN NORMAL AND VARICOSE VEINS

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INTRODUCTION

During the routine pathologic examination of surgical material it occurred to one of us (RLH) that very little information of value to the patient was being obtained from making microscopic sections of *varicose veins*, and that more information may be gained by simply opening up the segments of veins and counting the valves. After about a score of such specimens labeled "varicose veins" had been examined in this manner and each reported as *so many valves encountered in so many total centimeters of vein*—or *one valve per so many centimeters of vein*—the question arose as to what the *normal* ratio is. This phase of the problem was approached by examining the greater saphenous veins from sapheno-femoral junction to the knee in both lower extremities of the cadavers being dissected by the class in gross anatomy. This yielded data of a more exact nature that could be subjected to a variety of detailed analyses. Pertinent to the point, however, was the figure of *one valve per so many centimeters of vein* in *normal* veins.

When we had completed this preliminary study we were surprised to find from a perusal of the literature on the subject[2, 1, 3] that the possibilities of this very simple technic had not been exhausted, and that we were unable to find any figures for *varicose veins* comparable to those that we had accumulated. The only comparable data on *normal* veins that we have been able to find is that of Kampmeier and Birch.[2] Table 2 of their paper records the number and position of valves in the great saphenous vein in 32 extremities of 24 unclassified cadavers. The segment of this vein from the knee to the saphenofemoral junction averaged 42.3 cm. in length, and the average number of valves per segment was 6.4, or one valve per 6.6 cm. of vein. We have continued this study of *normal veins* for six additional years. This report compares the findings in 43 cases of *varicose veins* with those in the veins of 198 extremities of 102 *normal* individuals.

METHODS

The methods have to be described in some detail, for the validity and duplicability of the results hinge on this point. As there were difference between the handling of the *pathologic material* and the *normal material*, the methods applied to each will be described separately.

Pathologic Material: The 19 specimens examined during the first year consisted of segments of vein *strippings* removed in the operating room, wrapped in gauze moistened with saline, and delivered promptly thereafter to the labora-

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tory. They usually were examined within an hour or two after operation and always within 24 hours—in the cases with the longer intervals the specimens were kept in a refrigerator at 38 F. In other words this group of specimens was examined in the *fresh condition.*

The usual practice in the hospital is to send all tissue removed at operation to the laboratory, and the assumption is made that all the segments removed reached the laboratory. There was no way of knowing from the specimens submitted exactly which veins were removed, or whether the operation was done on one or both sides. There is not sufficient accurate clinical data available to estimate the degree of varicosity; and no attempt has been made to correlate the anatomic findings with the clinical findings (such as sex, age, family history, occupation, duration, and the presence or absence of secondary effects such as edema and ulceration). All the patients in Watts Hospital are white in contrast to the major source of *normal material.* The whole group was accepted as *varicose veins.*

The number of segments of vein per specimen varied from 1 to 9, and the length of the segments varied from 2 to 66 cm. Some of the segments of very small caliber were not opened because of technical difficulties, but it is reasonable to suppose that these were either tributaries or distal branches of the saphenous vein. All segments of 2 mm. or more inside diameter—in the *empty* state in which they were examined—were opened throughout their entire length, washed under running water, and the valves counted with the aid of a good light and a fine probe.

Counting of valves in *varicose veins* is not always an easy task as a number of dubious structures are sometimes encountered. Since this, in a sense, is the crux of the problem, it is necessary to describe these in some detail. Most of the textbooks of anatomy, embryology, and histology list unicuspid and tricuspid as well as the usual bicuspid valves in veins. Everything that fit any of those descriptions was counted as a valve. The principal difficulty encountered was what we have chosen to call *pseudo-valves*—structures that varied from slight thickenings, probably related to kinks, angulations, and tortuosities, to incompetent valves that closely approached normal. If there was any semblance of a cusp, i.e., if there was any semblance of a pouch that could be demonstrated by running water or careful probing, or if there was any definite *pairing* or other semblance of a commissure between what might have been cusps of a valve that had practically disappeared, the structure was counted as a valve. Even these criteria are sometimes difficult to apply, but an effort was made to count *all* valves in this group; and this was done before the *normal* figures became available. In this first group of 19 examined during the first year a total of 701 cm. of vein disclosed 52 valves, or an average of one valve per 13.5 cm. of vein.

The second group of 24 specimens was similar to the first except that they were examined after they had been fixed in 4 per cent solution of formaldehyde for 1 to 15 months. They were not as easy to dissect as the ones examined in the *fresh* state, nor was the recognition of valves in the opened segments as definite and complete. In this group the number of segments per specimen varied from 1 to 9, and the length of the segments varied from 3 to 65 cm. The total length

of vein examined was 1454 cm., and in this 76 valves—including all doubtful structures—were recognized—an average of 1 valve per 19.1 cm. of vein. When these two groups are combined, a figure of 16.8 cm. of vein per valve is obtained.

A few other possible pitfalls in the methods applied to the *pathologic material* should be mentioned. It is known that valves tend to occur at or near the junction of one vein with another. Conceivably this could be a source of error, for it would be possible always to start the operation just distal to a valve; but our surgeon friends have belittled this possibility. Another possible source of error which the surgeons consider more likely is that they might have cut through or ligated a vein at a valve, and this valve would not be included in the specimen sent to the laboratory, or might not be recognized as a valve if it were badly torn. All of these possible sources of error are admitted, but the most real one to us is the determination of *pseudo-valves*. In the interpretation of the results of these studies the obvious lengthening, tortuosity and dilatation that characterize varicose veins present by far the greatest obstacle.

Normal Material: The students in seven successive classes in gross anatomy cooperated in examining and recording the number and distribution of *competent valves* in 198 extremities of the 102 cadavers dissected during this period. All of these cadavers were assumed to be free from *varicose veins*. All were adult: 92 Negro and 10 white; 74 male and 28 female. In each extremity examined the great saphenous vein between the knee and the sapheno-femoral junction was opened and the total length, together with the number and position of all valves judged to be competent, was recorded. The majority by far of the valves were bicuspid, although occasional unicuspid and tricuspid structures were encountered. Localized thickenings of the intima which projected into the lumen and which simulated valves were recorded as *rudimentary* or *pseudo-valves*. These were *not* counted in computing the figures given in table 1.

The 198 veins examined varied in length from 20.5 cm. to 50 cm; the average length was 39.65 cm. The mean number of apparently competent valves found in these 198 veins was 4.5 $\pm$ 0.092 valves. The standard deviation of this mean was 1.93 $\pm$ 0.065. The range in number of valves found in all veins was 0 to 11. A competent valve was located at the sapheno-femoral junction in 89.5 per cent of the veins examined. Hesse and Schaack[1] have reported a similar figure of 87 per cent.

A total of 891 competent valves were recorded in 198 veins totalling 7852

TABLE 1

Valves in normal and varicose veins

Group	Number of Cases Examined	Total Length of Vein Examined	Total Number of Valves	Valve per cm. of Vein
Varicose				
a. Examined in fresh condition	19	701	52	13.5
b. Examined in fixed condition	24	1454	76	19.1
Total Varicose Veins	43	2155	128	16.8
Normal	198	7852	891	8.81

cm. in length. This is an average of 1 valve in 8.81 cm. of vein examined. It should be emphasized that this figure does not represent the average distance between valves.

Inspection of the data on the number of valves in the great saphenous vein of 1 thigh reveals a strong tendency to duplicate the number of valves in the vein of the opposite thigh of the same cadaver.

A summary of the pertinent normal and pathologic findings is presented in table 1. We cannot overemphasize the possible sources of error that have been described in detail under methods; all we can say is that every reasonable attempt has been made to avoid as many of these as possible. If we have been successful in this, the finding appears significant that a valve occurs on the average every 8.81 cm. in *normal veins* whereas *varicose veins* have only 1 valve to every 16.8 cm. of vein.

DISCUSSION

The significance which the above findings—namely 1 valve per 8.8 cm. of *normal vein* and 1 valve per 16.8 cm. of *varicose vein*—might have rests on a fundamental question: Does this ratio prevail at birth or is the *weakening* of the vein wall fundamental and the diminution in the number of valves a secondary consequence of this lengthening, tortuosity, opening up of additional unvalved channels? Until this question is answered any extended discussion is out of place. We may be pardoned for indulging in a little indirect logic and pointing out some of the fallacies in our current concept of the pathogenesis of varicosities, if only to stimulate someone to answer the fundamental question.

Most of the textbooks of pathology—and specific books on the subject of varicose veins[3]—list congenital weakness of the vein wall and increased pressure within the lumen as the principal causes of varicosity. This latter includes central—right-sided heart failure and cirrhosis of the liver—as well as local—tight garters, pressure of a tumor, pregnant uterus, or constipated rectum—factors, any or all of which may be aggravated by occupational habits exemplified by the clerk and the barber. But even in those cases in which these two conditions are fulfilled, it is the exception rather than the rule that varicosities develop, with the possible exception of cases of cirrhosis of the liver, and, if we exempt some of the posterior peritoneal veins and the occasional *caput Medusae*, these cases are better classified as collateral circulation rather than varicosity. Further we have been unable to find any accurate statistical evidence to substantiate the claim that varicosities are due to familial or congenital defects in the vein wall.

The congenital *defect* could as well be in the number and distribution of venous valves, and the weakening of the wall could be secondary to the increased pressure and the resulting stasis and anoxia. Certainly from a mechanical viewpoint a column of liquid—of specific gravity of 1.06–16.8 cm. high would exert more pressure on the vessel wall and be more likely to lead to incompetency of valves sooner than would a column of liquid only 8.8 cm. high.

The variation in the number and distribution of the valves within the group

assumed to be *normal* would lend some support to the idea that the number and distribution of valves at birth may be important factors in the subsequent development of varicosities. These factors could likewise operate in the hemorrhoidal and pampiniform plexuses as well as in the larynx, esophagus, bladder, and other sites where an isolated varix is sometimes found.

It is premature to speculate on the significance of this possibility; but anatomists, physiologists, pathologists, and clinicians alike well could ponder certain questions. What determines the number and distribution of valves in the venous system? Could one segment of vein devoid of valves over a sufficient length start the *vicious circle* that can be demonstrated by the Trendelenberg tests? What is this length of vein? Could a ligature or tight bandage applied at a critical point break this *vicious circle*? Or is the paucity and irregular distribution of valves too generalized? Does the success of the injection method for treating varicose veins depend on the frequent breaking of the *vicious circle* at one or more critical points by thrombus formation which finds favorable conditions at these points?

No proof is adduced here that the incompetence of valves preceded and did not follow the venous dilatation.

SUMMARY

Examination of 2155 cm. of vein included in 43 routine surgical specimens marked "varicose veins" disclosed 128 valves, or an average distance between valves of 16.8 cm.

Examination of 7852 cm. of vein in 198 extremities (from 102 cadavers) of *normal* individuals revealed 892 valves, or an average distance between valves of 8.8 cm.

The possible sources of error in the methods applied to this study and the possible significance of the results obtained are discussed.

CONCLUSION

This preliminary study suggests that the number and distribution of valves in the veins at birth may be a factor in the subsequent development of varicose veins.

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RETROPERITONEAL SEMINOMA (GERMINOMA)

REPORT OF A CASE. HISTOGENIC AND DIAGNOSTIC CONSIDERATIONS

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The extreme rarity of ectopic seminomas, if, indeed, they exist at all, combined with the apprehension with which a surgeon looks upon an abdominal mass the origin of which remains to be clarified, is the justification for the following case report, in which the diagnostic procedures are presented in some detail.

CASE REPORT

A white male school teacher, 37 years of age, was seen Feb. 2, 1952. A mass in the right side of the abdomen was present. It had first been observed late in December of the preceding year and had steadily increased in size, although at no time had it given rise to any symptoms.

Previous History. This patient was first seen April 4, 1936, at the age of 22, for a recurrent urethral discharge and mild discomfort after voiding not associated with frequency, urgency, or gross hematuria. In October 1934 he had contracted gonorrhea, for which he had been treated for seven weeks. He was then discharged as cured. No history of syphilis was obtained. At the age of 10 years a small tumor, described as a fibroma, was removed from the left breast, and about the same time he had an acute orchitis following parotitis. Sexually he was active and virile, with marked libido and normal orgasm. The family history was entirely negative.

Physical examination at this time (1936) was essentially negative except for a slight urethral discharge and rather small testes. The testis on the right appeared somewhat atrophied. Both were of normal consistency. The prostate was slightly enlarged and boggy, and a smear of the secretion revealed pus and a few gram-positive staphylococci. Microscopic examination of the serminal secretion revealed a marked oligospermia, but the spermatozoa were morphologically normal and active.

At this time, and at irregular intervals until 1950, the patient was treated for a recurrent nonspecific urethritis and chronic prostatis. When he next was seen, in February 1952, the only illness in the interim had been a recent so-called virus infection, characterized by headache, generalized pains, abdominal cramps, diarrhea and fever, for which he had been treated with aureomycin.

Physical Examination. Physical examination in February 1952 revealed a strong, healthy man, with the ruddy color typical of his race (Yugoslav). The general examination was negative. In the right side of the abdomen, extending from the flank mesially to beyond the midline, was a tense, ovoid mass, about 4 by 5 inches in its greatest extent, of somewhat limited mobility. Although the consistency was firm, it was the impression that the mass was cystic.

Diagnostic Procedure. Routine laboratory examinations were negative. Roentgenographic examination of the urinary tract (fig. 1) showed no urinary calculi, but the right kidney was pushed upward and outward by the mass just described. Its density confirmed the impression received on physical examination that it was cystic.

Intravenous pyelograms (fig. 2) showed the right kidney pushed slightly upward and

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FIG. 1

FIG. 2

FIG. 1. Kidney, ureter and bladder roentgenogram showing right kidney pushed upward and outward by large mass. The clarity with which the psoas margin and right kidney can be seen indicates that the mass is cystic.

FIG. 2. Intravenous pyelogram showing mass seen in figure 1 impinging on right kidney, causing displacement and rotation. On the left is a large double kidney, with double pelves and calices. The ureters are not visualized.

laterally, and the pelvis and calices slightly rotated. The left kidney was long and apparently double, with double pelves and calices. The ureters were not visualized.

Cystoscopy and retrograde pyelography (fig. 3) were negative except that the pelvis of the right kidney was slightly hydronephrotic. The kidney was displaced upward and slightly rotated. The right ureter was displaced laterally and apparently implanted on the posterior aspect of the renal pelvis. The pelvis of the left kidney appeared to be in the upper pole of a large kidney. It was agenic, as were the calices. Only one ureter was visualized on this side. Urine from both kidneys was clear. Sediment from urine from the right kidney revealed, under low power, occasional leukocytes and very occasional red blood cells. Stained sediment was free from bacteria. Indigocarmine given intravenously appeared in good concentration from the left kidney in three minutes and from the right in five minutes.

As only two ureteral orifices had been identified cystoscopically, even after the injection of indigo carmine, it was thought that there may be fusion of the ureters and that the abdominal mass on the right side may be due to hydronephrosis of a supernumerary kidney. Injection of the ureters and pelves by means of the Brash acorn tip ureteral catheter (fig. 4) revealed on the left side a double kidney with double pelvis and fusion of the ureter just before it entered the bladder. On the right side no fusion of the ureter could be demonstrated, nor could a supernumerary kidney be identified.

Examination of the large bowel by barium enema revealed no obstruction, gross filling defect, or other intrinsic abnormality. The ovoid mass in the right side of the abdomen was well demonstrated on the postevacuation film. It lay in the paravertebral line, extending from the level of the first to the level of the fifth lumbar disk. It exhibited sharp peripheral margination except at the upper pole, which was not distinct. It measured 15.5 by 6.5 cm. in its greatest projected vertical and transverse diameters. It did not originate in the intestine,

FIG. 3

FIG. 4

FIG. 3. Retrograde pyelogram showing displacement of right kidney by mass, as in figures 1 and 2. The rotation of the kidney is evidenced by the apparent posterior implantation of the ureter in the renal pelvis. On the left the catheter has entered one limb of a presumably fused ureter. The pelvis and calices are agenic.

FIG. 4. Pyelogram taken with Brash acorn ureteral catheter. On the right side the deformities observed in figures 1–3 are further accentuated by this technic. On the left, the ureter can be seen to fuse just before it enters the bladder.

nor did it affect it in any demonstrable fashion. The impression was that it lay below and medial to the visualized kidney. The roentgenologist stated that this impression could be confirmed or excluded by a review of the pyelographic studies, and that a similar review would be necessary to evaluate an apparent enlargement of the left kidney. He also stated that if a lesion of the urinary tract could be excluded, he would suggest visualization of the small bowel, to eliminate the possibility of intestinal tumor or duplication, and of tumor or cyst of the mesentery.

Roentgenograms taken after retroperitoneal insufflation revealed the left kidney to be normal in size, shape and position. The psoas margin was well outlined and apparently normal. The right kidney was displaced laterally and the lower pole extended outward, as if displaced by a mass. The air which enclosed it revealed it to be normal in size and shape. Medial to the lower pole (fig. 5) was a mass resembling a kidney in outline and extending from the transverse process of the second lumbar vertebra to the right sacro-iliac joint. This mass measured 13 by 7 cm. Various obliquities revealed that it lay in the same plane, and possibly in the same fascial layers, as the kidney. It was surrounded by air on all surfaces except the posterior. It displaced the kidney laterally and anteriorly. A smaller mass lay superior to this mass and medial to the upper pole of the kidney. This second mass, which measured 7 by 4 cm., extended from the lower margin of the body of the second lumbar vertebra on the right side. It was thought that it might be the right adrenal, which was not definitely identified. It was, however, larger than the normal adrenal gland would be and was not surrounded by air. It was not well visualized on the oblique or lateral views. Although these masses were retroperitoneal, they did not disturb the psoas margin shadow.

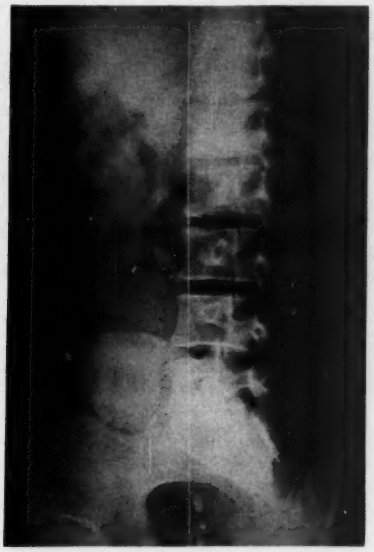

FIG. 5. Roentgenogram taken after retroperitoneal air insufflation. The retroperitoneal mass observed in figures 1–4 is excellently outlined.

Diagnostic suggestions were to rule out multiple kidney on the right; eliminate retroperitoneal tumors, such as lipoma, fibroma and sarcoma; consider the possibility of neurofibromatosis and investigate additional sites if clinical suspicion warranted. An incidental note was made of barium present in diverticula in the lower quadrants of the abdomen. Re-examination at 6 hours and 24 hours did not sharply define the upper mass. No other changes were noted.

When the patient was admitted to Touro Infirmary March 13, 1952, the physical examination was still essentially negative. There had been no evident change in the mass. The blood pressure was 140/80. A note was made that the testes were smaller than usual, especially on the right. The prostate felt normal in size and consistency.

Urinalysis, hematologic study, blood chemical determinations and blood serologic reactions revealed no abnormalities. The urinary sediment contained calcium oxalate crystals, a few epithelial cells, and 1 to 2 white blood cells and an occasional red blood cell per low power field. The intravenous phenolsulfonphthalein test was 70 per cent (60 per cent the first hour, 10 per cent the second). The Fishberg test was 1.030 for the first specimen and 1.033 for the second and third.

Operation. Operation was done March 15, 1952, under spinal analgesia, on a preoperative diagnosis of blocked hydronephrotic supernumerary kidney versus retroperitoneal cyst. The abdomen was opened through a transverse incision 2 inches below the umbilicus, extending from the midline to just beyond the semilunar line and then laterally and upward toward the costovertebral angle. The peritoneum, which was thin, was reflected mesially and to the left. It was torn twice (and repaired at once) before the retroperitoneal mass could be fully exposed. The mass was encapsulated and tense and gave the impression of being cystic. It was first thought that it was not adherent except at its point of attachment or origin on the nephrogenic ridge at about the level of the third and fourth lumbar vertebrae. When, however, it was being separated from its attachment, by sharp dissection, it became evident that it was adherent to the vena cava, in which a small nick was made. It

was readily repaired, but to avoid the risk of further damage to it, the capsule of the mass was incised and a trip was left in situ adherent to the vein.

It was noted, and commented upon, as the capsule was incised, that it was laminated. As the tumor was being delivered, the inner layer of the capsule ruptured and some of the contents were spilled. They consisted of clear fluid and pieces of soft, pinkish-white tissue suggestive of neoplastic tissue. The wound was closed in layers, without drainage.

Pathologic Report. Because of the unusual appearance of the tumor, an immediate frozen section was made and was reported as seminoma. As there had been no previous suspicion of such a diagnosis, no hormonal tests had been made. The morning after operation urine was collected and sent to the laboratory for an Aschheim-Zondek test, which was negative.

The complete pathologic report was as follows:

One specimen consists of four fragments of tissue, varying in size from 2.5 by 1 by 5 cm to 1 by 1 by 1 cm. Frozen section has already been made upon this specimen. The tissue is pinkish-white and soft in consistency, and there are multiple areas of local necrosis on the outer surface. This tissue has the appearance of sarcoma.

The second specimen consists of a mass measuring 6 by 10 by 5 cm. and weighs 182 Gm. The outer surface is hemorrhagic and injected. On section this specimen has the soft, watery consistency and pinkish-white appearance of a retroperitoneal sarcoma.

Microscopic examination (figs. 6 and 7) shows the tumor to be made up of large round or polyhedral cells with distinct cytoplasmic borders. The cells possess abundant clear cytoplasm, and large centrally placed hyperchromatic round nuclei with one or two prominent nucleoli. The cells are closely packed and the whole tumor has a rather homogeneous appearance. The stroma is scanty and contains small collections of small round cells re-

FIG. 6

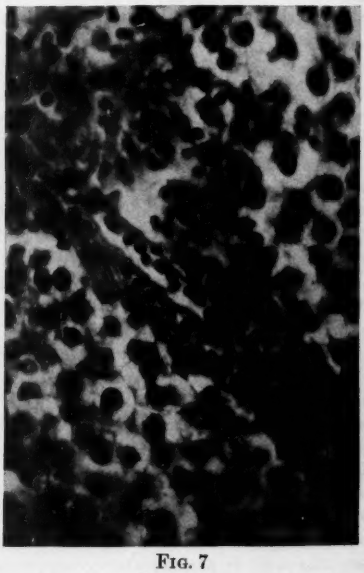

FIG. 7

FIG. 6. Photomicrograph (× 30) showing the outer margin of the retroperitoneal mass to be composed of condensed fibrous tissue. The uniform round cells composing the tumor are seen below.

FIG. 7. Photomicrograph (× 400) showing histologic structure compatible with seminoma: typical robust, generally uniform, round or polyhedral cells, divided into small alveolar groups by the fibrous stroma, in which can be observed the lymphoid component typical of this tumor.

sembling lymphocytes. Large dilated capillaries filled with red blood cells are seen in some areas within the stroma. One section shows a large area of necrosis. The tumor possesses a fairly thick fibrous capsule, which in some areas contains a portion of sympathetic chain and ganglia. Nothing resembling lymph node is seen. In some areas the tumor is seen to be invading the capsule.

The pathologist concluded that the histologic picture was indistinguishable from that of a metastatic seminoma of the testis. If a primary tumor of the testis could be ruled out, he thought the tumor could best be classified as a retroperitoneal sarcoma of nephrogenic ridge origin.

Postoperative Course. The patient's immediate postoperative course was entirely uneventful. When the sutures were removed March 22, several days after operation, the wound was clean and had healed by primary intention. Forty-eight hours later the patient complained of chest pain and had a slight elevation of temperature. The internist, who was called in consultation at once, made a clinical diagnosis of pneumonitis versus possible infarction. Roentgenograms of the chest substantiated the diagnosis of pneumonitis in the lower lobe of the left lung and in the right lung just above the diaphragm. Serial roentgenograms confirmed the satisfactory resolution of the pneumonitic process. Nothing in any of the roentgenograms suggested a metastatic neoplastic process. A complete laboratory work-up at this time, including blood cultures, was negative.

On April 1, 1952, the patient complained of pain in the upper left thigh and had a slight rise of temperature, this time as as the result of a mild thrombophlebitis.

He was discharged from the hospital in good condition on April 18, 1952, after completing a course of deep roentgen ray therapy (16 treatments in 19 days). Because of the pulmonary complications and thrombophlebitis, most of the treatment (4,682 r) was given through an anterior port and only 1,512 r through a posterior port. The skin reaction was minimal.

Follow-Up. The patient reported for examination at regular intervals after his discharge. His condition was consistently good, and he was able to resume all his usual activities. On October 16 and November 10, 1952, the Wiltberger-Miller test (Rana pipiens) was reported negative with concentrated urine.

The patient was in excellent condition, with no complaints, when he was examined in the middle of February 1953. When he was next seen, June 5, 1953, he complained of a painless induration under the skin below the surgical scar. It was not clear just when it had appeared. On examination, two superficial masses were found. One, about 1 inch in diameter, was just below the ensiform cartilage, and the other and larger one, about 4 by 2 by 2½ inches, was about 4 inches below the umbilicus. Both masses were firm, nodular, and freely movable. Examination of the urine by the modified Wiltberger-Miller frog test was negative.

Although it was considered highly improbable that these masses were metastatic, excision and biopsy were done under spinal analgesia on June 20, 1953. Only the larger mass was excised. It was yellowish and indurated. It was not attached or adherent to the sheath of the rectus muscle. It was regarded as a response to roentgen therapy. The wound was closed with drainage.

No preceptible drainage was observed, and when the sutures were removed on the eighth postoperative day, healing seemed satisfactory. Two days later the skin edges separated for about 3 inches. This area was left open to heal by granulation. The patient was discharged in good condition on July 12, 1953. He has remained well to the date of writing (Dec. 1, 1953).

The pathologic report was as follows:

The specimen consists of an elliptical portion of skin measuring 11 by 5.5 cm. The skin presents a hemorrhagic discoloration and is somewhat roughened. A large amount of subcutaneous fat measuring 3 cm. in thickness is attached to it. The fat is indurated, and some areas of pale yellow color suggest fat necrosis. There is no evidence of localized tumor masses. The remainder of the specimen consists of soft adipose tissue. Microscopic examination of numerous sections shows only fat necrosis; there is nothing suspicious of neoplasm.

COMMENT

Pure seminoma (germinoma) of the testicle is an uncommon tumor. According to Friedman and Moore,[7] who studied 922 tumors of the testicle sent to the Army Institute of Pathology between October 1940 and May 1946, 35 per cent were pure seminomas. Terato-carcinoma accounted for another 35 per cent of the specimens, embryonal carcinoma for 19 per cent, teratoma for 7 per cent, and miscellaneous tumors for 3 per cent. Most of the patients were of military age (18–38) and were serving in the United States Army. The group from which the tumors were obtained was therefore relatively homogeneous.

Hinman[8] cites Southam and Linnell's observation that these is one tumor of the testis for every 1,500 male surgical admissions to general hospitals; what percentage of these would be seminomas is not stated.

A review of the records of Charity Hospital of Louisiana at New Orleans for the 10 year period ending in December 1952 shows that 126,836 males were admitted to the institution, of whom 64,091 were white and 62,745 were negroes. Pure seminoma of the testicle was encountered in 9 patients, 4 between the ages of 27 and 39 years, and 4 between the ages of 44 and 51 years. The remaining patient was 81 years of age. Eight of the patients were white, and one, 27 years of age, was a Negro. This is a disproportionate racial incidence, but the numbers are too small to permit any conclusions.

Only 3 cases of pure seminoma have been observed in a private urologic practice covering 48 years. All were white men, who were between 35 and 46 years of age. Although the number of patients, again, is too small to permit conclusions, the age incidence in both private practice and at the Charity Hospital supports Friedman and Moore's observation that while the majority of these tumors occur during the years of greatest sexual activity, the incidence increases with advancing years. The condition, according to Campbell,[3] is practically unknown in infancy and childhood.

According to Friedman and Moore, the characteristics of seminomas can be summarized as follows:

1. They respect tissue planes as they grow, as well as adjacent structures, and are not aggressively invasive or destructive as are embryonal carcinomas.

2. Invasion of the adnexa and spermatic cord is exceptional.

3. The small incidence of regional and distant metastases, like the low mortality rate, is consistent with the relatively benign appearance of these tumors.

4. Metastases are usually restricted to retroperitoneal tissues or peritoneal linings. Distant and parenchymal metastases are very uncommon.

5. The architecture of testicular tissue is not reproduced in seminomas and the neoplastic cells do not resemble spermatogonia. Since they are probably tumors of primordial germ cells they should be called germinomas.[5, 6, 7]

6. Seminomas are distinct biologic entities. For the most part they are pure mononuclear growths, devoid of teratoid structure, no matter what stage of development is observed.

7. All the evidence is thus to the effect that seminomas are more benign than the embryonal carcinomas and teratoid tumors.

The resemblance between seminoma cells and some elements in seminiferous tubules, Friedman and Moore point out, has been taken as evidence that seminomas arise from the cells (spermatogonia) of the testicular tubules themselves. Small seminomas in the early stage of development incidentally found in the hilus of the testicle at autopsy and studied by serial sections have yielded no evidence to substantiate the theory of tubular origin. The possibility that seminomas (germinomas may represent neoplasms of the so-called primordial germ cells should be investigated.

Because of the apparent absence of a testicular tumor in the case reported in this communication, the histologic diagnosis of seminoma was made with some hesitancy. For that reason slides were submitted to Colonel J. E. Ash,[1] Consultant in Pathology, United States Army, Retired, who stated that he did not see how the growth could be classified as anything but a seminoma. It was made up of typical robust, generally uniform, round or polyhedral cells, divided into small alveolar groups by the fibrous stroma, in which was present the lymphoid component typical of this tumor. Its location may suggest a neural origin, but there was no other evidence to this effect. The histogenesis of the tumor, Colonel Ash concluded, was difficult to explain; extragenital seminomas have been observed, and it was theoretically possible that this particular tumor arose locally from a rest of the primary gonadal element. Without further knowledge of the condition of the testis, no more could be said.

At Colonel Ash's suggestion, Dr. Nathan B. Friedman,[4] Cedars of Lebanon Hospital, Los Angeles, was also asked for his opinion. After examining the specimens, he stated that in his opinion the tumor was unquestionably a germinoma (seminoma), the best possibility being that it arose in the testis and that serial sections of the testicle from the same side would reveal a tumor focus or a residual scar. A less likely possibility was that it had arisen in accessory testicular tissue along the urogenital ridge, as suggested in the original pathologic report. A growth in the retroperitoneum, he thought, must be called metastatic until both testes had been serially sectioned, and either tumor or scar ruled out. He regarded the writer's suggestion that the tumor may be primary, in view of the structure of the capsule, as a possibility, but pointed out that a lymph node completely replaced by neoplastic tissue would present the same appearance.

In a study of 23 neoplasms of the pineal gland collected at the Army Institute of Pathology from civilian and military sources, Friedman[5] found that many so-called pinealomas are identical with certain tumors of the testicle. In his opinion, 10 of these 23 specimens could be classified as germinomas, as he prefers to call seminomas, on the basis of their probable origin in primodial germ cells.

A search of the literature reveals little else that is helpful. In 1942 Bazy and Denoix[2] reported a retropancreatic tumor considered a seminoma, but without an apparent primary lesion in the testis. The patient, a 44 year old man, was seen in December 1941, with a mass in the upper abdomen, to the left of the rectus abdominis, and extending to the edge of the costal margin. It had been painful for the last four months. He had lost a great deal of weight during the last eight months. Except for a small gastric ulcer on the lesser curvature of the

stomach and mild distress attributable to it, the patient was otherwise asymptomatic. Study of the function of the pancreas and of the gastric secretion was not helpful. On Jan. 3, 1942, the abdomen was opened by a midline incision. Exploration revealed a large retroperitoneal mass, over the surface of which coursed enormous blood vessels. This mass apparently was adherent to the vertebral column and its removal was regarded as impossible. When the thin capsule was incised in an avascular area, to secure a specimen for examination, a pinkish pap exuded. This substance occupied the center of the tumor. The interior tissues were whitish, of uniform consistency, and yellowish bodies were observed, like those in an ovary. As much as possible of the interior of the tumor was extirpated and the resulting cavity was drained.

Histologic examination of the specimen showed that the mass was a seminoma. No abnormalities of the testes could be found. They were normally situated in the scrotum and were of normal size and consistency.

Surgery was followed by deep roentgen ray therapy. The site of the drain formed a persistent draining sinus, which took months to close. The patient was in excellent condition; had regained his strength and presented no abnormalities of the testes during the period of observation, which covered 10 months after operation.

The author concluded his report by observing that until there was evidence of pathologic changes in the testis, the origin of the tumor must remain in doubt. Was it a metastasis, or did it arise from an embryonic remnant of the genital ridge?

In discussing this case, Huet recalled a somewhat similar patient upon whom Lecène operated. No lesion of the testis was evident at the time. Sauvé also recalled an analogous case, on Launay's service, at which he had assisted. The abdomen of this patient was opened because the diagnosis of pancreatic tumor had been made and an enormous, inoperable tumor of the para-aortic lymph nodes was found. Several weeks later the origin and nature of the tumor were clarified at autopsy, after section of the right testicle revealed a very small seminoma.

Whether or not the seminoma (germinoma) reported in this communication is primary, originating from the urogenital ridge, seems debatable, in view of Friedman's[6] observation that the original testicular tumor, after becoming metastatic, may undergo involution to the point of not being recognizable except by the multiple block section technic. As Sauvé's case shows, apparently normal gonads may be the site of primary seminoma. Clinically, there was every reason to believe that his case was a pancreatic tumor, and the true nature of the neoplasm was revealed only at autopsy. Bazy and Denoix's case and Huet's case, like my own, must remain obscure until the testicles can be sectioned, an obvious impossibility under the circumstances in all 3 cases.

There are several points in my own case which suggest that the tumor might be a primary rather than a metastatic seminoma:

1. The capsule was thick and laminated. Against this observation must be cited Friedman's[4] comment that a completely destroyed lymph node may present the same appearance.

2. The tumor arose from the nephrogenic ridge and there was no evidence at operation of enlarged para-aortic nodes. It would be rather unusual, if this huge tumor were a metastasis, to find no other regional nodes involved.

3. It would also seem unusual that a metastasis could occur in a node remote from the original tumor without some involvement of the intermediate nodes.

4. The pathologist stated that sympathetic chain and ganglia were observed in some areas of the capsule. He also stated that nothing resembling a lymph node could be identified.

If the tumor were a primary seminoma, the possibility is advanced that it might have arisen from primary germ cells in the nephrogenic ridge. If so, it would seem logical, as Friedman[5] states in his discussion of pinealomas identical with certain tumors of the testicle, to describe it as a germinoma rather than a seminoma.

Diagnostic Considerations. In 15 of 29 presumably primary retroperitoneal teratoid tumors submitted to the Armed Forces Institute of Pathology between 1942 and 1947, the retroperitoneal mass was the first evidence of a testicular tumor.[6] In 6 cases either the testes had not been examined or routine testicular sections showed nothing. In 8 cases significant lesions were present in the original sections of the homolateral testis or were found by examining all available testicular tissue by the multiple-block method. In 1 of these 8 cases a small but definite tumor undergoing necrosis and fibrous encapsulation was found by studying additional sections after routine autopsy sections had disclosed nothing of significance. Friedman concluded that so many retroperitoneal teratoid tumors have proved to be metastases of primary genital tumors that one questions whether primary teratoid growths of the retroperitoneum actually exist. Certainly the diagnosis of extragenital chorioepithelioma in man cannot be accepted incontrovertibly unless the testes have been carefully studied and can be shown to contain neither neoplastic nor cicatrizing lesions.

As a rule, the differentiation of an intraperitoneal mass from a mass in the retroperitoneal space is not difficult. When the mass is accompanied by pain, the character of the pain, its point of origin, and its localized or referred nature are all of diagnostic value. When, as in the present case, the mass is not painful, it is more difficult to determine its precise location, but physical examination, instrumental investigation and roentgenologic studies are usually conclusive. When the mass is retroperitoneal, it may be possible to determine its precise nature before exploration.

As Hinman pointed out, well over 25 years ago, a retroperitoneal mass should be considered of renal origin until it is proved otherwise, in view of the fact that renal masses far outnumber nonrenal masses in this location. In the differential diagnosis of renal from nonrenal masses, valuable information can be secured, as in this case, by complete urologic studies, including perirenal air insufflation. Angiography is often of value in obscure cases.

After renal conditions have been excluded, there still remains the problem of determining whether the mass is adrenal, parietal, or unattached, and whether it arises from urogenital embryonic remnants or is a metastatic spread from unrecognized testicular, ovarian or other neoplasia. By correlation of the evidence

obtained by all possible investigations with the clinical syndrome present, it may be possible to differentiate further between cortical and medullary tumors of the adrenal glands. In other nonrenal retroperitoneal masses, a preoperative differential diagnosis usually is impossible.

SUMMARY

A case is reported in which a retroperitoneal mass, symptomless except for its presence, was removed at exploratory laparotomy, undertaken, after exhaustive investigation, on the incorrect diagnosis of blocked hydronephrotic supernumerary kidney versus retroperitoneal cyst. The histologic diagnosis, concurred in by several outstanding pathologists, was seminoma. No evidence of a tumor of the testis was found at this time, nor has any evidence appeared in the 20 months which have elapsed since operation.

The origin of seminomas is discussed, and it is suggested, tentatively, that this tumor may represent an instance of ectopic seminoma, arising from primordial germ cells, though it is realized that this hypothesis cannot be established in a still young, virile man. The diagnostic considerations of retroperitoneal masses are also discussed.

Note. Appreciation is expressed to the pathologists who have so kindly interested themselves in this case, Dr. A. J. Herzog, Pathologist, Touro Infirmary; Dr. Russell L. Holman, Professor of Pathology, Louisiana State University School of Medicine; Colonel J. E. Ash, Consultant Pathologist, United States Army, Retired; and Dr. Nathan B. Friedman, Cedars of Lebanon Hospital, Los Angeles, formerly of the Army (Armed Forces) Institute of Pathology.

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ACUTE CHOLECYSTITIS FOLLOWING UNRELATED SURGERY

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It is disconcerting to be faced with the problem of acute abdominal distress in a patient who has recently had an operation. It is understandable that a complication of the operation usually is considered if the operation was an abdominal one, and if a nonabdominal operation preceded the symptoms, then blame is centered on the disturbed gastrointestinal motility that occurs after anesthesia. This has caused difficulty in making the diagnosis of acute cholecystitis in the postoperative period, and has led to delay in instituting proper therapy.

In 1947 Glenn[1] called our attention to this topic when he presented 17 patients in whom acute cholecystitis followed a surgical procedure unrelated to the biliary tract. More recently, Sparkman[4] presented a paper on abdominal emergencies following unrelated surgical procedures, in which the 6 cases of acute cholecystitis constituted one-half of the total number reported. All of these had associated cholelithiasis. Schwegman and DeMuth[3] reported on 17 patients observed in the Hospital of the University of Pennsylvania over a 23 year period. Apparently the occurrence of acute cholecystitis in the postoperative patient is not as uncommon as the paucity of cases in the literature would suggest. The recognition of this condition is difficult, but of vital importance.

Glenn's explanation for the occurrence of acute cholecystitis in the postoperative period is as follows: For a number of hours before the operation the patient receives nothing by mouth. Morphine and atropine are given. It is believed that the flow of bile to the gallbladder is relatively constant and is not influenced by these drugs. Bile is being concentrated in the gallbladder and there is no stimulation of the gallbladder to contract until the patient is given a soft diet. At this time there is contraction of the gallbladder which attempts to empty itself of thick, viscid concentrated bile. If a stone blocks the cystic duct, the mechanism for the development of obstructive cholecystitis becomes obvious. In his experience there were a few instances of acute cholecystitis without cholelithiasis.

Experimental support of this thesis was presented by Thomas and Womack.[5] In normal dogs they found that withholding food for 48 hours caused concentration of bile in the gallbladder. If, at this time, obstruction of the cystic duct were created surgically, acute cholecystitis developed. This did not occur if the period of fasting did not precede the cystic duct ligation. The concentration of bile was relatively normal, i.e., 6.5 Gm. per cent of cholic acid and 174.9 mg. per cent of cholesterol in those dogs fasting for 48 hours, compared

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to 4.9 Gm. per cent of cholic acid and 147 mg. per cent of cholesterol in the nonfasting animal. This re-emphasizes the role of stasis of concentrated bile and obstruction in acute cholecystitis.

The present series was taken from the files of Touro Infirmary in New Orleans from 1933 through 1952. During this time, 21 patients were found who developed acute cholecystitis following operations unrelated to the biliary tract. A number of factors were considered in this study to see if a common denominator existed, or if any of the theories of the etiology of acute cholecystitis could be supported. The importance of the subject was quite evident when one noted the confusion and delay caused by the onset of acute cholecystitis in the postoperative period.

AGE, SEX AND DISTRIBUTION

The distribution over the years was fairly constant, occurring about once or twice a year since 1940. There were 14 men and 7 women, a reversal of the expected predominance of females in cholecystic disease. The youngest patient was 34 years of age and the oldest was 83, with an average of 57.6 years. Fifteen of the 21 patients were over 50 years of age (tables I and II).

OPERATIVE PROCEDURES

The operative procedures that were done prior to the onset of acute cholecystitis may be seen in table III. There were 6 urological cases, and multiple procedures were performed in a few patients. It is obvious that the type of operation preceding the onset of symptoms of acute cholecystitis has no relationship to gallbladder disease. Seven of the operations were on the extremities, back, or face, and all of the urologic procedures were extraperitoneal in nature.

DRUGS

Most of the patients received preoperative medication. In 1 patient this is not known, and 1 patient had no preoperative medication. Morphine and

TABLE I

Sex	Number	Percentage
Males	14	66.7
Females	7	33.3
Total	21	100.0

TABLE II

Age	Number	
30–39	3	
40–49	3	
50–59	6	
60–69	3	15 over age of 50
70–79	4	
80–89	2	
Total	21	

TABLE III

Operative Procedure	*Number*
Inguinal hernioplasty	3
Transurethral prostatic resection	2
Suprapubic cystostomy	2
Nephrectomy	2
Appendectomy	2
Cataract extraction	1
Amputation of the lower extremity	1
Partial gastrectomy	1
Skin graft to hand	1
Ileotransverse colostomy	1
Splenectomy	1
Laminectomy for ruptured intervertebral disc	1
Radical mastectomy	1
Incision and drainage of abscess of hand	1
Resection of carcinoma of nose	1
Liver biopsy	1
Hysterectomy	1

atropine were the drugs used most frequently, while 2 received dilaudid and atropine, and 1 scopolomine (table IV).

PREVIOUS SYMPTOMS

Eight of the patients had symptoms of gallbladder disease in their histories. A negative history was recorded in 8 patients, and no mention of such symptoms was made in 5. A more careful history might have indicated symptoms suggestive of gallbladder disease. This is important especially when considering operation in a patient over the age of 50. If one can prove that cholelithiasis is present before the contemplated surgery is done, one can give credence to symptoms of acute cholecystitis should they occur in the postoperative period.

CHOLELITHIASIS

The presence of cholelithiasis is very important. The theory that obstruction of the cystic duct is necessary for the development of acute cholecystitis is becoming more evident. Of the 21 patients, stones were present in the gallbladder in 8, absent in 6, and their presence was not determined in 7 patients. The

TABLE IV

Preoperative Medication	*Number*
Morphine	17
Dilaudid	2
Atropine	19
Scopolamine	2
None	1
Unknown	1

TABLE V

Cholelithiasis	Number	Percentage
Present	8	38.0
Absent	6	28.6
Unknown	7	33.4

fact that not all patients had stones causes one to look for other causes of cystic duct obstruction (table V).

FASTING PERIOD

The effect of solid food, i.e., soft diet, has already been noted. In the present series there were 2 patients who received no solid food before the onset of acute cholecystitis, and in 2 patients it is unknown whether or not they had solid food. The remainder of the patients were placed on a soft diet on the average of 2.7 days postoperatively. This ranged from the first to the seventh postoperative day, and fits fairly well with the experimental observations of a 48 hour fasting period. The time of the onset of the attack following the ingestion of food is as follows: in 2 patients this was not known, in 4 there was no solid food ingested, and in the remainder the time interval varied from 1 to 16 days. The average number of days from the time of ingestion of food to the onset of the attack was *4.2 days* if one excludes the 2 patients whose intervals were 14 and 16 days.

The length of time after the operation that the attack of cholecystitis began is a combination of the above figures and averaged 9.8 days. The shortest interval was 6 hours and the longest was 33 days, and was unknown in 2 patients.

ANESTHETIC

The type of anesthetic given was not a factor, since 7 of the patients had spinal anesthesia, 2 had local infiltration of ovocaine, and 10 had general anesthetics (table VI).

TABLE VI

Anesthesia (many patients received multiple agents)	*Number*
Spinal	
Pontocaine	1
Novocaine	4
Nupercaine	2
General	
Pentothal	6
Ethylene	7
Ether	9
Cyclopropane	5
Nitrous oxide	2
Local	
Novocaine	2

TABLE VII

Treatment	*Number*
Cholecystectomy	6
Cholecystostomy	4
None	9
Proved at autopsy or not treated at operation	2
Total	21

DELAY

Once the patient had an onset of symptoms of acute cholecystitis, the fact that previous surgery recently had been done led to delay in diagnosis and therapy. At times the delay was intentional, but misdiagnoses occurred, especially in those patients who had had a laparotomy. There was no delay in 6 patients, an intentional delay in 2 patients, and in the remainder the delay averaged *2.5 days*. In 8 patients there was a diagnosis other than acute cholecystitis in the postoperative period, e.g., subdiaphragmatic abscess, acute appendicitis.

TREATMENT

The treatment of these patients was conservative in 9, cholecystectomy was done in 6, and cholecystostomy was done in 4 (table VII). There were 2 patients in whom cholecystitis occurred as a terminal event and the diagnosis was made at autopsy. There were 3 deaths in the series, none due to cholecystitis.

CASE REPORTS

The following case reports will illustrate some of the difficulties encountered.

Case 1. S. D., a 57 year old woman was admitted to Touro Infirmary on Sept. 15, 1949 with a diagnosis of diabetic gangrene of the left foot. On September 20, an amputation of the left leg was done under pentothal, curare, and nitrous oxide anesthesia. She was allowed a soft diet the day after operation and the onset of cholecystitis occurred on the third postoperative day, with vomiting, mild, cramping abdominal pains, and distention. Two days after the onset of symptoms a diagnosis of acute appendicitis with perforation was made and the patient was taken back to the operating room. At operation, the gallbladder was found to be edematous, tense, and the tip showed gangrene. A cholecystectomy was done. Stones were present in the gallbladder. There was a delay of two days before operation in this patient, and retrospectively a history of previous attacks of cholecystitis was obtained.

Case 2. E. T., a 72 year old man was admitted to Touro Infirmary on July 2, 1951 with a diagnosis of intestinal obstruction probably due to carcinoma of the cecum. Six hours after admission an ileotransverse colostomy was done under spinal anesthesia. Because there was a large carcinoma of the cecum completely obstructing the terminal ileum, a two-stage resection was planned. He was placed on a soft diet on the seventh postoperative day and discharged on the eleventh postoperative day. Two days later he had an onset of nausea, vomiting, and abdominal cramps after ingesting a very heavy meal. This was six days after being placed on a soft diet. On readmission flat and erect roentgenograms of the abdomen revealed distended loops of small bowel with fluid levels, and no gas was seen in the colon. A diagnosis of intestinal obstruction due to malfunctioning of the ileocolonic

stoma was made. A Miller-Abbott tube was passed; the patient rehydrated and after three days of preparation with saline enemas, he was taken to surgery. Because of the rather poor risk the patient presented, this plan was adopted rather than subject him to two surgical procedures. At operation, the Miller-Abbott tube was found to be through the ileocolonic stoma, which was obviously patent. The gallbladder was found to be tense, edematous, and thickened, and showed subsiding acute cholecystitis. It was left intact and a right colon resection was done. The patient died of carcinomatosis one year later. During this time no symptoms of cholecystitis appeared.

Case 3. L. W., a 53 year old woman was admitted to Touro Infirmary on Aug. 14, 1946 with a diagnosis of infected hydronephrosis on the right side. On August 15, a right nephrectomy was done under cyclopropane and ether anesthesia. She was given soft food on August 17, the second postoperative day, and on August 26, nine days later, she developed pain in the right upper quadrant. This had been preceded by two or three days of mild nausea. A previous gallbladder visualization showed a poorly functioning gallbladder, and because the signs and symptoms were not severe, delay occurred. Five days after the onset of symptoms she was operated upon as an emergency and a very edematous friable gallbladder found. A cholecystectomy was done and no stones were found.

Case 4. R. B., a 61 year old man was admitted to Touro Infirmary on Nov. 10, 1949 with a diagnosis of chronic gastric ulcer. On November 14 a partial gastrectomy was done under pentothal, ethylene and ether anesthesia. The patient was allowed solid food on the fourth postoperative day, and on the seventh postoperative day he had persistent mild nausea and vomiting. On the ninth postoperative day fever and leukocytosis were present. A mass was palpated in the right upper quadrant. The next day he was taken to the operating room with a preoperative diagnosis of a subhepatic abscess and perforation of the duodenal stump. At exploration the stump of the duodenum was found to be intact, and the gallbladder was thickened, edematous, and gangrenous. No stones were present, and a cholecystectomy was done.

DISCUSSION

A study of this type does not pretend to settle the debate as to the etiology of acute cholecystitis. Rather, it serves to emphasize the factors that may culminate in a serious condition in the immediate postoperative period. As Maes[2] points out, ". . . the patient who suffers a surgical accident while in the hospital, undergoing treatment and surrounded by all the resources of medical science, is actually more likely to lose his life than is the patient who is stricken in his home or on the street." The importance of the early discovery of acute cholecystitis has been emphasized by many authors who strongly urge immediate cholecystectomy. In the early postoperative period, credence must be given to symptoms of colicky epigastric pain, nausea, vomiting, fever, and tenderness in the right subcostal area, especially when the symptoms occur after a fasting period of 2 to 4 days. The presence of cholelithiasis will further serve to make one suspicious, *but the absence of gallstones by previous examination should not lull the surgeon into a sense of security.* In this series only 38 per cent of the patients had gallstones present. However, in over 33 per cent, the presence of cholelithiasis was not determined.

The sex distribution is of interest since there is a reversal of the expected female predominance with two-thirds being males. This is in agreement with the figures of Schwegman and DeMuth.[3]

The types of operation preceding the onset of acute cholecystitis were of a

large variety, the only significant factor being that there were 6 urological cases. This would be expected in considering the age group that is involved.

The question of the effects of preoperative medication is one which cannot be evaluated, since almost all patients receive a narcotic with either atropine or scopolamine. It is to be noted that in at least 1 patient, no preoperative medication was given. Glenn believes that these drugs play an insignificant role in the genesis of acute cholecystitis in the postoperative period.

A factor of considerable importance is the relationship of ingestion of food to the onset of the acute attack. This interval ranged from 1 to 16 days, with over half of the cases occurring within one week after the ingestion. It could not be ascertained from the charts as to what *type* of food was eaten, and the effect of a restricted diet may explain the long delay in some of these cases.

SUMMARY

From this study, no conclusions as to the etiology of acute cholecystitis can be drawn. It should be emphasized that acute cholecystitis does occur in the postoperative period, especially in men over the age of 50. It is the most common cause of acute abdominal disease in the interval after operation, other than those complications related to the surgery. This fact also strengthens the plea of many surgeons, of strongly recommending cholecystectomy in patients with so-called silent gallstones.

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LYMPHANGIOMA

ESSENTIAL DATA IN FORTY-TWO CASES FROM CHARITY HOSPITAL OF LOUISIANA AT NEW ORLEANS

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Lymphangioma, a neoplasm of lymphatic tissue, is an extremely uncommon disease which is widely distributed throughout the body. It must be distinguished from lymphatic varix, which is merely a dilated lymphatic. In view of the enormous regenerative capacity of the lymphatics,[4] it is surprising that lymphangiomas are not more common. There still is no general agreement concerning their origin and etiology.

Lymphangiomas will not be conceived of correctly unless the function of the lymphatic system is understood. In man, the lymphatic circulation is rudimentary and a variety of forces combine to transport the lymph through the channels, all of which are valved. These forces include arterial pulsations, muscular activity, intestinal peristalsis, and alterations in the intra-abdominal and intrathoracic pressure. Visceral, parietal and peripheral lymph eventually collects and blends in the cisterna chyli, to be evacuated via the thoracic duct.[16] The chief lymphatic ducts are connected by a maze of interlacing collaterals, and as a result, when lymphatic obstruction occurs, the lymph circulation may become retrograde and paradoxical.[4]

The role of lymphatics in the body economy is primarily the removal of excess blood protein from the tissues. Derangement of this function results in lymph stasis and edema.[16]

Lymphangiomas do not receive a great deal of attention in the medical literature. The purpose of this communication is to review the recent important articles on the subject and to present the essential data, with brief comments, in 42 consecutive cases of lymphangioma treated at a single institution, Charity Hospital of Louisiana at New Orleans.

REVIEW OF THE LITERATURE

As the literature indicates, lymphangiomas may be localized or diffuse. The diffuse variety may involve one or more extremities in their entirety. In 1953 Freeman[10] reported 8 such cases. The majority of visceral lymphangiomas have been reported as individual cases, and no statement concerning their incidence is possible. Cystic hygromas, a special variety which appears in the neck, axilla, groin, or, very occasionally, the retroperitoneal tissues,[29] are practically always discussed under the heading of lymphangioma.

In addition to their appearance in the extremities, the axilla,[23] and the neck,[8, 26, 28] lymphangiomas have also been reported in the kidney,[31] spermatic

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cord,[21] scrotum,[9] uterus,[14] vagina,[19] intestine,[5, 11, 15, 20, 22, 30] spleen,[25] omentum,[1, 2, 12] mesentery,[7, 17, 18] bone,[3, 13] larynx,[6] and tongue.[24]

Only two large series of lymphangiomas have been reported in the recent literature. Singleton,[27] in 1937, reported 28 cases. The report included no instances of visceral lymphangioma and 16 of the 28 neoplasms were located in the head and neck. Watson and McCarthy,[29] in 1940, reported 41 cases of lymphangioma and 14 of cystic hygroma. The lymphangiomas were distributed in the head and neck (52 per cent), extremities (44 per cent) and trunk (4 per cent). None was visceral. Of the 14 hygromas, 13 were in the neck and 1 in the axilla. Up to 1940, according to these observers, 225 cases of hygroma had been reported in the literature. Dowd's[8] collection, in 1913, included 126 cases. In contrast to the relative frequency of cystic hygromas of the neck Watson and McCarthy noted that only 2 per cent of all other types of lymphangioma were located in this region.

ANALYSIS OF DATA

Forty-two proved cases of lymphangioma appear in the records of Charity Hospital of Louisiana at New Orleans up to Jan. 31, 1953; the first was recorded in September 1935. One patient had two neoplasms. The essential data are presented in tabular form (table I), but certain points deserve special comment.

The extreme infrequency of these neoplasms is obvious from the period of time over which these 42 cases occurred. Nineteen cases occurred after 1949, and the largest number treated in a single year, 7, were observed in 1950. The small size of the series permits no general conclusions, but it should be noted that 4 cases were observed in 1938 and 4 in 1942. This suggests that increasing awareness of the disease probably plays no great part in the larger numbers observed in recent years and that the yearly distribution is entirely accidental.

In 30 of the 42 cases the age of onset was under 15 years. Twelve patients were under 1 year of age, and 4 of these were under 1 month. In 1 instance the diagnosis was made on the second day of life. At the other extreme, 1 patient was 76 years of age. The youth of the patients is typical. Twenty-six of Singleton's patients presented the lesion at birth or in early childhood. The same age incidence was observed in 95 per cent of Watson and McCarthy's 41 lymphangiomas and in 11 of their 14 cystic hygromas.

The sex incidence of 26 males and 16 females needs no special comment. Of particular interest, however, is the fact that 29 of the patients were Negroes. The race is seldom mentioned in reported series, but Charity Hospital is an institution which has always had a large Negro population; at the present time it is approximately 60 per cent. Singleton mentioned that 2 of his patients were Negroes, and Watson and McCarthy stated that there was 1 Negro in their 14 patients with cystic hygroma. By inference, all 41 of their patients with lymphangioma were white.

Thirty-seven of the lymphangiomas in the Charity Hospital series were parietal, 10 in the head, 17 in the neck, 4 in the upper extremity and 6 in the

TABLE I

*Essential data in 42 consecutive cases of lymphangioma**

Case no.	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21
Year	35	37	38	38	38	38	39	39	39	41	41	42	42	42	42		43	44	44	45	45
Age	30Y	1M	12Y	1Y	4Y	9Y	16Y	51Y	15Y	2W	42Y	18Y	16Y	12W	68Y	9M	6W	13Y	12Y	1Y	6Y
Race-sex	NF	NM	NM	NF	WhM	WhM	NF	NM	NM	WhM	NM	NM	NF	WhM	NF	WhM	WhF	NM	NM	NF	NF
Location																					
Head						xR												x	x		
Neck	xR	xL			xL		xR		xR				x			x				xR	
Parotid																					
Submaxillary G															xL						
Upper E			xR													x					xR
Lower E				xL						xL		xR				x	x				
Liver											x										
Intestine								x													
Mesentery														x							
Pathology																					
Duration	6M	1M	2M	1Y	4M	1Y	2Y		4Y	2W		17Y	18M	6M	1Y	9M	6W	11Y	7Y	1Y	6Y
Subcutaneous	x	x	x	x	x	x	x		x	x		x	x			x	x			x	x
Submucosal								x					x	x				x	x		
Simple	x				x	x		x			x				x					x	x
Cavernous																	x	x	x		
Cystic		x		x			x		x				x	x							
Infected			x									x					x		x		
Sclerosing																					
Diffuse																x					
Soft	x	x		x		x	x	x	x					x		x		x		x	x
Hard									x	x	x			x			x		x		
Diameter (cm.)	4	6	1	8.5	1.2	15		2.9	5	4	0.3	25	6	18.5		11	12		3		8
Treatment and results																					
Excision	x	x	x	x	x	x	x	x	x	x		x	x	x	x		x		x	x	x
Death													x								
Recurrence						x			x								x				

Case no.	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38	39	40	41	42
Year	46	47	49	49	49	49	50	50	50	50	50	50	50	51	51	51	51	52	52	52	53
Age	76Y	25Y	3M	16Y	6Y	50Y	5Y	9M	3W	14M	2Y	64Y	28Y	33Y	2Y	6M	44Y	4Y	32Y	15M	4D
Race-sex	NM	NM	WhF	NF	NM	NM	NM	WhF	NF	NF	NF	NM	WhM	WhM	NF	WhM	WhM	WhM	NF	NM	NM
Location																					
Head					xL	xL	xL	x					x	x					xL		
Neck	xL		xL	xR						xR					xR	xR	xL	xR			xR
Parotid																					xR
Submaxillary G																					
Upper E											xL										
Lower E		xR							xR												
Liver																					
Intestine												x									
Mesentery																					
Pathology																					
Duration	9Y	1M	3M	1½Y	1Y	2Y	1¼M	9M	3W	14M	24Y		47Y	74Y	24Y	6M	9M	2W	1M	3M	4D
Subcutaneous	x	x	x	x	x	x			x	x	x			x	x	x	x	x			x
Submucosal							x	x					x	x		x			x		
Simple	x				x	x			x			x							x		
Cavernous								x			x				x						
Cystic			x	x												x	x	x		x	x
Infected							x			x			x						x		
Sclerosing		x												x							
Diffuse																					
Soft	x		x	x	x		x								x	x	x			x	x
Hard								x	x	x	x		x	x				x			
Diameter (cm.)	9	1	12	12	3.5	4	1	1.7	10	2.4	5.2	2.5	0.6	8	4	5.5	10	8.0	2		5.5
Treatment and results																					
Excision	x	x	x	x	x	x		x	x	x	x	x	x	x	x	x	x	x	x	x	x
Death								x	x			x									
Recurrence		x	x	x				x			x							x			

* Key: W = weeks. M = months. Y = years. D = days. N = Negro. Wh = white. M = male. F = female. E = extremity. R = right L = left. G = gland. Diameter = greatest diameter in centimeters.

lower. In 1 instance there was diffuse involvement of both the upper and lower extremities.

Of the 6 visceral lymphangiomas, 1 was in the parotid gland and 1 in a submaxillary gland. Three of the others were incidental findings. The lymphangioma of the liver (case 11), apparently the first to be reported, was found at autopsy. The lymphangioma of the mesentery (case 14) was recognized at an emergency operation for intestinal strangulation. The lymphangioma of the jejunum (case 33) was recognized at a gastric resection for duodenal ulcer. It presented as five small polypoid masses, which were resected. The single visceral lymphangioma recognized and resected deliberately was in the rectum (case 8). It was diagnosed by proctoscopic biopsy section.

From the standpoint of pathologic process, 27 lymphangiomas were subcutaneous, including the neoplasm of the parotid gland (case 42). Eight were submucosal, including the neoplasms of the rectum and the mesentery. Three were both subcutaneous and submucosal. The consistency was soft in 22 cases and hard in 13. In the remaining cases this point was not mentioned. The diameter ranged from 0.3 cm., in the lesion in the liver, to 25 cm., in a lesion in the lower extremity.

There was no instance of malignant degeneration in this series during the period of observation. There was also no instance of hemorrhage at operation or any other time. Hemorrhage, infection and fistula formation are common complications of the neoplasm itself and of its treatment. Eight lymphangiomas in the Charity Hospital series were infected, and 2 (cases 12 and 17) were complicated by fistula formation. Infection can be an extremely serious complication, particularly when operation is done, because of the abnormalities of the lymphatic system. Lymphangioma is also a common cause of lymph fistula. Twelve of the 40 cases reported from the Mayo Clinic in 1949[16] were of this origin.

Three patients had no treatment in the hospital, in 1 instance because, as already noted, the neoplasm which was in the liver, was an autopsy finding. One patient refused surgery and in the other patient, an instance of diffuse involvement, surgery was not considered practical. Irradiation therapy was not used in any case. It is the method of choice, for obvious reasons, in diffuse systemic lymphangioma, but the results are not impressive. As Watson and McCarthy point out, the dose, to be effective, must be so heavy that scarring results. Irradiation therapy was used in 18 of their 41 patients; in 25 per cent the results were poor, and in 35 per cent the growths recurred.

Recurrence is frequent, whatever the method of treatment. Seventeen of the 41 cases observed by McCarthy and Watson were instances of recurrence, after treatment elsewhere, and their personal incidence of recurrence was 12 per cent. Four lymphangiomas recurred in the 13 patients treated by wide excision. The tendency to recurrence, they note, apparently increases with age.

In the Charity Hospital series there were 9 known instances of recurrence after excision. The actual number probably is much higher, since no attempt at formal follow-up of these patients was made.

The death of the patient with a lymphangioma of the liver was, as already

noted, unrelated to the growth. Similarly, the death which occurred in the case of lymphangioma of the jejunum was properly attributed to the gastric resection at which time it was found. One patient died from a fall after excision of a lymphangioma of the head. Death occurred shortly after operation, but was in no way related to it. The two other deaths were related to the therapeutic procedure. One patient died of atelectasis after resection of a lymphangioma of the neck and another of shock after resection of a lymphangioma of the leg.

SUMMARY

Lymphangioma is an uncommon neoplasm widely distributed throughout the body. Visceral growths are particularly infrequent. The literature on the subject is scanty. The important articles in the recent literature have been reviewed, and the essential data in 42 cases observed over an approximately 18 year period at Charity Hospital of Louisiana at New Orleans have been presented and commented on.

Although no instance of malignancy was noted in this series, lymphangiomas are characteristically invasive and poorly defined.

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A NEW TECHNIC FOR THE STUDY OF STRANGULATION OBSTRUCTION

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The study of intestinal obstruction is complicated by a multiplicity of variables. Elimination of some of the variables, or subdivision of the subject to permit more accurate study has made possible some of the major advances in the past. These have been based on:

1. The separation of simple and strangulation obstructions.[7]
2. The response of simple obstruction to proper fluid and electrolyte control alone.[6]
3. The role of shock and blood loss in the strangulation of long segments.[5, 8]
4. The ability of antibiotics to prolong life in strangulation obstruction.[1, 2, 3, 4]

Even with all these advances survival remains limited in strangulation obstruction. Chart 1 reviews the variables still encountered and indicates the value of reducing the variables to be studied.

In evolving a new method for the study of strangulation obstruction, selective control of the contents of the strangulated segment has been obtained for the first time. This should aid in evaluating the cause of death since recent evidence has indicated that the *lethal agent* is first formed in the strangulated segment.[10, 11] The role of each of the elements normally present in a strangulated, obstructed bowel (Chart 2) can be better evaluated through individual study in the strangulated segment described here.

TECHNIC

Vaccinated, medium size dogs are deprived of food for 18 to 24 hours, and water for 4 to 6 hours prior to operation. Sterile technic is used throughout. Under intravenous nembutal anesthesia a midline abdominal incision is made. The omentum is removed to prevent its causing too many adhesions, and to prevent its providing blood supply to the strangulated loop following the second operation. The small bowel is divided 75 per cent of the distance from the ligament of Treitz to the cecum (fig. 1). This permits a standard relationship for all dogs. The bowel again is divided 45 to 60 cm. proximal to this point. An end to end anastomosis of proximal and distal bowel is made, using a single layer of continuous silk suture. The mesentery of the two segments is brought together to prevent herniation. The proximal end of the remaining bowel loop is closed with a Parker-Kerr suture. The distal end of the isolated loop is brought out as an ileostomy either through the incision or a stab wound. A modified Furniss clamp is left on the exteriorized portion of the ileostomy until it sloughs off. This prevents wound contamination and permits the loop to become adherent

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Changes in Strangulation Obstruction

Irritation of parietal peritoneum	Electrolyte imbalance
Oral intake stops	Hemorrhage in bowel wall, lumen
Negative nitrogen balance	Anoxia of bowel wall, lumen
Normal intestinal transit stops	Bacteria proliferate
Stasis in obstructed segment	Bowel permeability increases
Bowel distends	Death of bowel
Vomiting and dehydration	Shock

CHART 1

Strangulated Loop Contents

Gastric, duodenal, small bowel secretions
Bile
Activated pancreatic juice
Blood
Bacteria
Sloughing mucosa

CHART 2

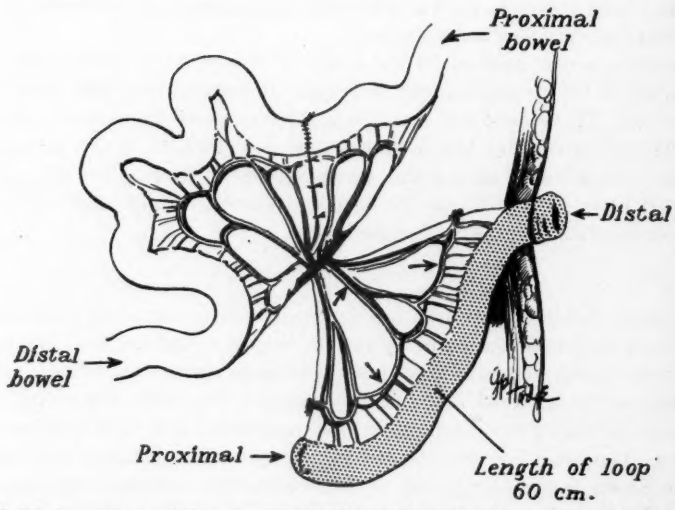

Fig. 1. Diagram of operative procedure. End to end anastomosis and closure of mesentery shown. Isolated loop with proximal end closed and distal end exteriorized. Arrows indicate points where veins are divided and ligated at second operation, to give a 30 cm. strangulated segment.

without necessitating sutures in the bowel wall. Once the clamp has fallen off, the dog cleans the stoma and surrounding skin, so there is no skin excoriation.

Following operation, the dog is given only intravenous fluids for 48 hours, oral liquids for the next 48 hours, and then regulation kennel diet. A period of 2 to 4 months allows return to preoperative weight. This long interval permits the isolated loop to evacuate any gastrointestinal contents present at the original

operation. Biliary, pancreatic, gastric, or duodenal secretions present in the loop would be expelled by its secretory flow and peristaltic activity. The loop then contains only its own secretions or any substances introduced for experimental purposes. In the interval between operations any substance under study may be periodically introduced into the loop. Absorption from an isolated segment of small bowel may be studied and the effect of any agent on subsequent strangulation can be evaluated. Since normal peristaltic activity expels substances placed in the loop, loops should have the ileostomy at the proximal end if it is desirable to keep injected substances in the loop.

The second operation is done 2 to 4 months after recovery from the first procedure. Through the old incision, the previously isolated loop is identified and its length measured. Points are selected so that a 30 cm. segment can be completely deprived of its venous supply by isolating, dividing, and ligating all veins in the mesentery of this segment. Arteries and veins parallel to the bowel at each end of the 30 cm. segment are divided and ligated to give a 30 cm. isolated, strangulated, but not closed loop of small bowel. Following strangulation any substance under study may be placed into the loop for study of its effects. Rubber tubes, placed in the peritoneal cavity and brought out through stab wounds, allow peritoneal fluid to be aspirated and studied during the survival period.

Of 12 dogs subjected to the first operation, 4 have survived. Four failed to survive because they pulled at the stoma and succeeded in pulling the entire loop out of the abdomen. One died of gangrene of the loop. Even autopsy has not revealed the cause of death of the remaining 3 animals. Results of the second operation will be reported at a later date. The technic appears to have sufficient usefulness to warrant an early report of the method so that it may be tested elsewhere.

DISCUSSION

This technic has eliminated a number of the variables listed in Chart 1.

Since the second operation does not in any way interfere with the continuity of the gastrointestinal tract and since the animal eats after recovery from anesthesia, there is:

1. No cessation of oral intake.
2. No negative nitrogen balance.
3. No cessation of normal intestinal transit.
4. Stasis and distention in the strangulated loop need not occur since the ileostomy continues to be an accessible outlet.
5. Vomiting and dehydration would occur only as a result of absorption of some noxious agent from the loop since there is no obstruction to the continuity of the gastrointestinal tract.
6. Electrolyte imbalance will not occur in the absence of vomiting, and with a normal intake.
7. Bacterial proliferation in the loop could be controlled by the instillation of antibiotics into the loop.

Factors *not* controlled by this technic are:

1. Irritation of parietal peritoneum. This has never been considered a major cause of death and need not cause undue concern.

2. Hemorrhage and anoxia of the bowel wall and lumen. This will occur as long as venous obstruction is present. If the length of the strangulated loop is properly chosen (30 cm. as used here) blood loss can be adequately replaced.

With conditions controlled as outlined with this technic, further advances should be possible in the study of strangulation obstruction. It now becomes possible to study selectively any one substance, or any combination of substances, in an isolated and strangulated loop. Absorption may be compared in a normal vs. a strangulated loop, since formation of a noxious substance is unimportant if absorption does not occur. Different substances may be placed in the strangulated loop to see if they alter the speed of the lethal process. These studies should make possible a more accurate appraisal of the toxin producing agents.

Murphy and Brooks briefly utilized a similar condition in the study of *simple* obstruction.[9] Their method was lost sight of, presumably because other factors soon proved of more importance in simple obstruction, and no other workers seem to have used this technic in the study of intestinal obstruction. The differences between simple and strangulation obstruction need not be elaborated here, but the ideas of Murphy and Brooks regarding the importance of *toxins* in simple obstruction are now known to be erroneous. It is surprising that no one previously has utilized this technic in the study of *strangulation* obstruction since it appears to be so ideally fitted to this problem by virtue of its ability to eliminate many of the undesirable side effects of strangulation obstruction. When the method was first utilized here, it was thought to be original, and it was only after a careful search of the literature revealed the paper by Murphy and Brooks that it became apparent that a similar technic had been used in the study of *any* phase of intestinal obstruction.

SUMMARY

A new method has been described for the study of strangulation obstruction, significantly reducing the number of variables to be evaluated. The method permits control of the contents of the strangulated segment, something not achieved before. More accurate study of the factors producing a toxin in strangulation obstruction is thereby made possible.

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ADDENDUM

Since this paper was completed, two additional papers have come to our attention in which a similar type preparation was utilized. Both were studies of *simple* obstruction, and both were studies of distention as a major factor in the cause of death in simple obstruction.

Herrin, R. C. and Meek, W. J.: Distention as a factor in intestinal obstruction, Arch. Int. Med., *51*: 152, January, 1933.

Taylor, N. B., Weld, C. B., and Harrison, G. K.: Experimental intestinal obstruction, Canad. M. A. J., *29*: 227, September, 1933.

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ASPIRATION DURING ANESTHESIA AND SURGERY

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The aspiration of vomitus is one of the great hazards of general anesthesia. When vomiting and regurgitation are massive, death swiftly follows from asphyxia; when less profuse, pneumonitis, suppuration or pulmonary edema follows. In certain instances the quantity regurgitated is small, and occurs silently. Most of the time the anesthetist is not even aware that it has occurred. There is little that the seasoned anesthetist fears more than the patient with a full stomach who begins to vomit during or as he emerges from anesthesia. Once vomiting occurs there is little that can be done to prevent aspiration. Prophylaxis is better than cure in these situations.

The terms *regurgitation*, *vomiting* and *aspiration* are used loosely and often interchangeably by many clinicians; however there is a difference between them. Vomiting, although it is largely involuntary, requires some voluntary effort for completion of the act. The recti muscles contract and help expel the gastric contents by compressing the stomach against the posterior abdominal wall. Reverse peristalsis in the stomach and the esophagus helps propel the gastric contents upward. The muscles of the pharynx, the fauces and the tongue become rigid and assist in propelling the material outward into the mouth. Vomiting occurs when the skeletal muscles are still under central control, that is, during the first and second stages of anesthesia. It cannot occur during the third stage because, in this state, voluntary efforts are abolished and superficial reflexes are obtunded. An alert anesthetist is aware of vomiting occurring during general anesthesia because swallowing and retching and spasmodic movements are apparent. Regurgitation, on the other hand, is devoid of the manifestations of striated muscle activity. It occurs without warning; most often it is silent, and it is usually associated with water collections in the gastrointestinal tract. It is usually precipitated or influenced by positional changes, manipulations of the viscera, particularly of the stomach, and anesthesia deep enough to relax the cardiac sphincter. The term aspiration indicates that the regurgitated material has passed into the respiratory passages by gravitation or by being drawn inward by the inspiratory negative pressure.

The question of aspiration is an important one. Recently the writers were intrigued by two reports—a preliminary one by Weiss (2) and the final one by Culver, Makel and Beecher[1]—which stressed aspiration during anesthesia and particularly emphasized silent regurgitation. These writers introduced a water soluble dye into the stomach preoperatively and attempted to recover it from the pharynx and trachea at the conclusion of operation. Their studies included observations on 300 surgical patients, 285 of whom were given nitrous

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oxide-ether anesthesia. Interestingly they noted an incidence of silent regurgitation of 25 per cent of the total in their series. Some of their findings are at variance with current impressions concerning regurgitation. For instance, one startling finding was that regurgitation occurred more frequently in the inclined or head-down position than in the supine. Inasmuch as this is the position in which patients expected to vomit or regurgitate are placed, it would be desirable to know whether or not this observation is correct. If it is, we certainly must revise our thinking and procedure in this respect. In view of the fact that there is a paucity of data on this subject, it is most important that these findings be verified. The report was largely confined to the use of nitrous oxide-ether anesthesia. It was our belief, therefore, that the study should be repeated and should embody a diversified series of cases to include all types of operations and a miscellaneous group of anesthetic drugs and technics.

METHOD OF STUDY

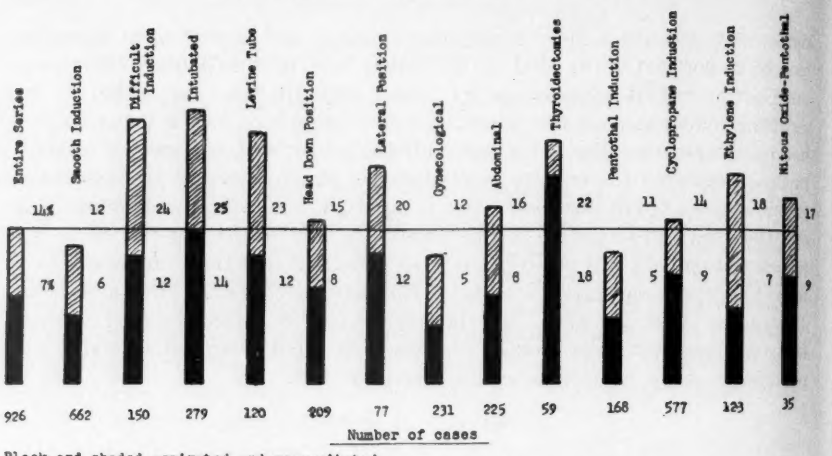
The methods used in our study were essentially similar to those of the authors cited above. Instead of a water soluble dye we used an insoluble one—carmine red. This obviated the possibility of false negative results due to absorption of the dye from the stomach and by false positives due to its absorption and excretion into the mucous and saliva of the upper respiratory tract. At the conclusion of anesthesia the pharynx was inspected by the aid of a laryngoscope and the contents of the trachea were aspirated. The carmine red was identified by adding ammonia water to the secretions aspirated. The ammonia causes the dye to dissolve and a deep red color appears. Blood turns brown in the presence of ammonia due to the formation of hematin. Thus the dye easily can be differentiated from blood.

RESULTS

A total of 926 patients were studied. Their ages ranged from 10 to 90 years. All types of operative procedures were included except those having oral or intra-pharyngeal surgery (Chart 1).

Fourteen per cent of the entire group regurgitated. Seven per cent, or roughly one-half of the group who regurgitated, aspirated. Not all patients who regurgitate, aspirate. Except during attempted intubation the anesthetist was not aware that regurgitation had occurred until the operation was over.

Relationship of Smoothness of Anesthesia to Regurgitation. Anesthetists are aware of the fact that smoothness of induction and maintenance of anesthesia are directly concerned with retching, vomiting and aspiration. Allowing the patient to become light during maintenance or inability to quickly pass through the second stage during induction predisposes to vomiting, retching, regurgitation and aspiration. Twelve per cent of the total designated as having an uneventful induction and maintenance regurgitated. Of these 6 per cent aspirated. This is approximately the average for the group as a whole. One hundred and fifty anesthesias were designated as difficult by the anesthetists. Twenty-four per cent of these regurgitated and 13 per cent aspirated. This is almost twice the



Black and shaded aspirated and regurgitated
Black only-aspirated

CHART 1

frequency of the average for the entire group. Obviously, then, smoothness of induction and maintenance plays a distinct role in the incidence of aspiration. Inasmuch as smoothness of induction is closely linked with the skill of the anesthetist, it can be inferred that the less skillful will encounter this complication more frequently. This we have found to be so in our series since our service is largely manned by student personnel and comparisons easily may be made.

Influence of Endotracheal Intubation. In view of the fact that endotracheal intubation is closely related to smoothness of induction and maintenance of anesthesia, and that patients may emerge into the lighter planes of anesthesia during or immediately after intubation, the incidence of regurgitation during this manipulation was studied. Twenty-six per cent of the total number of cases studied were intubated. Of the total intubated 24 per cent regurgitated and of this group half of them aspirated. Thus the incidence of regurgitation and aspiration is twice the average for the entire series. The intubations were oral without a cuff. Approximately half had packs introduced; the remainder were without packs. In certain instances regurgitation occurred at the time of intubation, in others sometime later. Exactly when regurgitation occurred was not determined because the inspection was made at the conclusion of anesthesia. The presence of the pack did not prevent aspiration when regurgitation occurred. The presence of the tube likewise was no assurance against aspiration. Presumably the gastric contents passed around the endotracheal catheter.

Influence of Stomach Tubes. Many have believed that the presence of a stomach tube insured against regurgitation and aspiration. Others have considered that its presence permits the cardiac sphincter to remain patent and relaxed and favors regurgitation around the tube. Approximately 12 per cent of the total number in the series were anesthetized with a Levine tube in situ. Of these 21 per cent regurgitated and 12 per cent aspirated. The incidence of aspiration

and regurgitation is almost double the general average for the series. There are several reasons which may explain this increased incidence. The presence of the Levine tube does not permit the anesthetist to apply the mask snugly to the patient's face. From a technical standpoint this makes the induction and maintenance of anesthesia more difficult. Then also, the stomach tube is used for patients who have distention or gastric retention more often than not. The possibility of vomiting is greater in this group because of the gastrointestinal disturbance. These findings suggest that the proper procedure would be to empty the stomach by aspiration, withdraw the tube and reintroduce it after the operation is completed.

Influence of the Position of the Patient. The position of the patient plays a role in aspiration. For years it has been accepted practice to place the patient in the head-down position when vomiting and regurgitation were anticipated. Beecher and Weiss noted the incidence of regurgitation to be greater in the head-down position in their series. Approximately 21 per cent of the patients in our series were placed in the head-down position with varying degrees of steepness. Fifteen per cent of these regurgitated and 8 per cent aspirated. The incidence of regurgitation and aspiration thus differs little from that of the general average. The majority of patients in the head-down position were having gynecologic operations. The incidence of regurgitation after gynecologic operations was 12 per cent and of aspiration 5 per cent. This is slightly less than the average. When abdominal surgery was done 16 per cent regurgitated and 8 per cent aspirated. These findings are in variance with those of Beecher and Weiss. It appears that the head-down position has no appreciable if any effect upon the incidence of regurgitation.

The dictum that water cannot flow uphill—referring to the fact that if a patient is inclined head-down he cannot aspirate—is thus not sustained by both our findings and those of Beecher and Weiss. Although it is true that water cannot flow uphill, it can be sucked up irrespective of the tilt of the patient. The regurgitated or vomited material lodges in the hypopharynx and initiates a spasm of the larynx in these circumstances. As the spasm is released a sudden, quick inspiration occurs. Whatever foreign material is present in the vicinity of the glottis is drawn upward into the trachea. The writers are familiar with a number of fatalities in which solid food was drawn into the trachea even though the patient was steeply inclined.

The lateral position is more conducive to vomiting, regurgitation and aspiration than the prone or supine. Eight per cent of the total operations were done in the lateral position. Of these 20 per cent regurgitated and 10 per cent aspirated. The incidence obviously is above that of the average for the entire series. We are at a loss to explain this. Perhaps the pressure of the kidney rest compresses the midportion of the trunk and compresses the stomach. The head-up and foot-down, or Fowler's position, frequently is employed for thyroidectomies. Six per cent of the patients were operated upon in this position. Twenty-two per cent regurgitated and 18 per cent aspirated. These were done entirely with endotracheal anesthesia which, as has been pointed out previously, carries a

considerably higher incidence of aspiration than the average. This is the only group in which the incidence of regurgitation and aspiration approximated each other. In other instances noted the incidence of aspiration has been about half that of regurgitation. Obviously the head-up position favors aspiration. The data does not support Beecher's contention that the head-up position is preferred to the head-down to obviate regurgitation and aspiration.

The Relationship of the Type of Anesthetic to Aspiration. The type of anesthetic used has an important bearing on the smoothness and ease not only of induction of anesthesia but maintenance as well. It is common knowledge that once an anesthetic is complicated by retching, obstruction, and other factors it may remain so for the duration of the operation. Cyclopropane and pentothal are used to attempt to secure a smooth uneventful induction for ether anesthesia. Smooth induction of anesthesia using nitrous oxide-ether sequence is difficult to secure and requires skill. In this series cyclopropane was used for induction in 57 per cent, pentothal in 17 per cent and ethylene in 13 per cent of the cases. The incidence of smooth inductions was with cyclopropane, 82 per cent, with pentothal 84 per cent and with ethylene 72 per cent. The incidence of regurgitation and aspiration for cyclopropane and pentothal paralleled each other. Eleven per cent regurgitated and 5 per cent aspirated when pentothal was used, while 14 per cent regurgitated and 7 per cent aspirated when cyclopropane was used. Eighteen per cent regurgitated and 7 per cent aspirated when ethylene was used for induction. The nitrous oxide-pentothal combination was used from the beginning to the end of operation. Three and one-half per cent of the total number of cases were done using this technic. Of these 90 per cent were reported as having smooth inductions. The incidence of regurgitation was 17 per cent and aspiration 9 per cent. This is higher than the average.

The combination of ethylene and cyclopropane was used for induction in 4 per cent of the cases. The fact that the two gases were combined may be taken as presumptive evidence that difficulty was experienced with ethylene, and cyclopropane, which is a more potent drug, was added to facilitate the induction. This is attested to by the fact that 70 per cent of the inductions were reported as smooth and 30 per cent as difficult. The incidence of regurgitation was 20 per cent while 18 per cent aspirated. It thus can be seen that an uneventful induction may be secured using cyclopropane or pentothal, and that in so doing the incidence of regurgitation and aspiration may be minimized. Our findings—using ethylene preliminary to ether—fall in line with those of Beecher for nitrous oxide ether. Both ethylene and nitrous oxide are pharmacologically similar and are used for identical purposes.

DISCUSSION

Morbidity, surprisingly, was absent in patients who aspirated. There are several explanations for this. The quantity of material regurgitated could not be estimated. However, in every instance it was small. All patients known to have aspirated were placed on a regime of antibiotics. They were followed closely during the postoperative period and observations, including pulse rate, respira-

tory rate, percussion, auscultation and a roentgenographic examination of the chest were made for the first few days. No evidence of pneumonitis or bronchitis was detected.

Relationship of Regurgitation and Aspiration to Anesthetics Other Than General. The misconception that regurgitation is possible only under general anesthesia is widespread. The writers are familiar with a number of instances in which vomiting followed by aspiration, which caused immediate fatalities, occurred while local infiltration, nerve block or spinal anesthesia were being used. Often regional anesthesia is selected for poor risk patients. Many of these are desperately ill, and semicomatose with an obtunded cough reflex. Lying supine with the arms fastened to the sides, these patients are unable to help themselves when they vomit or regurgitate and they aspirate vomitus from the first wave of the antiperistaltic rush. The argument that a conscious patient cannot aspirate because his cough reflex is active is far from correct. Any patient in the supine position will readily aspirate should he vomit, because he cannot quickly extricate himself from it and sit up. During operation almost all patients are in this situation.

Patients with full stomachs, who ordinarily are not prone to vomit, invariably become nauseated and vomit when they undergo the manipulations of surgery. The barbiturates, including pentothal in sedative amounts, allay nausea. However, when barbiturates are used for surgical anesthesia regurgitation occurs after—if not during—the surgical procedure, if the patient has a full stomach.

A question which is often asked is what should be done in the event a patient has ingested a meal, particularly of solid food? It is well known that in normal individuals the stomach empties within four or five hours. In apprehensive patients—in those who are ill, or in those who are under emotional stress—this is not necessarily true. The writers have seen undigested food regurgitated 12 hours after it was ingested. How long a patient should be allowed to wait for operation after ingestion of a meal is a question which is difficult to answer because of the vast differences in emotional makeup of each individual. Certainly elective surgery should be deferred for at least 24 hours after ingestion of food. The operation should be postponed as long as possible when emergency surgery is indicated. In the event operation must be done the anesthetist must be prepared to cope with the vomiting at all times, but more particularly at the conclusion of anesthesia. A skillful anesthetist anesthetizes the patient as quickly as possible and maintains anesthesia at an even keel. If perchance the patient is allowed to become light at anytime, vomiting may occur during the procedure, often with catastrophic consequences. Emetics administered preoperatively are, as a rule, not satisfactory. The patient may vomit once or twice after an emetic but this does not necessarily assure one that the stomach has been completely evacuated. In addition, certain emetics, notably apomorphine, have a depressant effect which may be objectionable in conjunction with the anesthetic.

The suggestion has been made that the stomach should be lavaged to accomplish evacuation. Gastric lavage is an ordeal which vexes the patient. More often than not the discomfort of the procedure often outweighs the advantage

gained. Anyone who has had experience with gastric lavage knows that seldom is the lavage fluid which was introduced into the stomach totally recovered. In the writers' experience lavage has made the situation worse rather than better. We believe that lavage not only is not indicated, but is contraindicated. The safest course to follow is to defer operation as long as possible under these circumstances.

SUMMARY

This is a study of aspiration during anesthesia made in 926 patients who were given an oral dye preoperatively. The dye was identified in the pharynx and trachea by laryngoscopy.

Aspiration of gastric contents during operation may be massive and is followed by asphyxia, pneumonitis, or abscess formation. Regurgitation during general anesthesia may occur silently and may be unknown to the anesthetist. This silent regurgitation occurs more often when difficulty is encountered with induction and maintenance of anesthesia. The incidence is higher in patients who are anesthetized with a stomach tube in place and in patients who have intratracheal anesthesia. The incidence of regurgitation in the head-down position is average; in the lateral position it is greater than average. The incidence, when anesthetics such as cyclopropane or pentothal are used, was less than with nitrous oxide or ethylene ether. The former permit smoother, more rapid induction of anesthesia.

The skill of the anesthetist plays an important role in the problem.

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OCCULT CARCINOMA OF THE BREAST

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Occult carcinoma of the breast is a term used to describe a cancer of the breast without any clinical evidence of a mass in the mammary gland. The first manifestation of this condition is distant metastasis to the axillary or supraclavicular lymph nodes on the affected side; to viscera, or to bone.

The purpose of this report is to review briefly the literature on this condition; to emphasize its clinical aspects and to report 6 cases that have come to our attention in the past three years on the surgical service at Touro Infirmary in New Orleans, Louisiana.

A total of 48 cases of occult carcinoma of the breast—proved at operation—have been reported in the literature. However, some of these cases do not fit our criteria for this condition and therefore are not included in this report. Several authors have classified as occult carcinoma of the breast patients in whom the presenting sign was a bloody discharge from the nipple, although no mass was present in the breast. One reported a patient with edema of the skin of the breast, nipple retraction and a Pagetoid appearance of the areola of the breast without a demonstrable mass. According to the strict definition of occult carcinoma, there should be nothing on or in the breast to indicate that an underlying mass is present.

Occult carcinoma has been called *hidden carcinoma of the breast, silent carcinoma of the breast, latent carcinoma of the breast,* and *axillary carcinoma of the breast* without a demonstrable mass. We believe that the most logical term for this condition is occult carcinoma, for, as this name implies, it is hidden from sight and palpation. In this sense it is similar to occult blood in the stool; while the blood cannot actually be seen in the patient's feces, it can be detected by laboratory examination.

This manifestation of cancer is not confined to the breast alone. Metastatic spread may be the first sign of malignancy in thyroid cancer, clinically known as aberrant thyroid. Skeletal metastases may be the first clinical manifestation of prostatic, thyroid, or renal carcinoma. The primary growth cannot always be found when squamous cell carcinoma has metastasized to the cervical lymph nodes. Nevertheless, in all of these conditions, a primary tumor exists, and, if the patient lives long enough, sooner or later it will show itself. Fortunately, it is not always necessary to wait for this to happen, for, with excision biopsy of a metastatic lesion, the surgeon may be given a direct clue to the site of the primary growth.

HISTORY

This entity is by no means new. Halsted[5] in 1907 reported 4 cases of this condition. Three patients had enlarged axillary lymph nodes without a detect-

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TABLE I

Cases of occult carcinoma of the breast

Reported by	Age	Race	Sex	Symptoms	Duration of Symptoms	Survival after Surgery
Blackburn in Cogswell's paper	60	W	F	Axillary mass	Not given	13 years and living
Cameron	?	W	F	Lump in armpit	Unknown; removed yr. before	1 year follow-up
	Elderly	W	F	Tumor of armpit	Several months	3 years
	56	W	F	Swelling of axilla, redness, oozing	2 months	Not given
Cogswell	45	W	F	Mass in left axilla, pain down left arm	6 weeks	Not given
Coleman in Cogswell's paper	50	W	F	Mass in neck below left ear	Not given	6½ years and well
Davis in Cogswell's paper	75	W	F	Mass in right axilla	6 months	5 years and living
Fitts and Horn	35	W	F	Lump in axilla; aching in axilla radiating to breast 1 year before	2 weeks	4 years and well
	46	W	F	Lump in left armpit	2 months	3 years and well
	63	C	F	Lump in axilla	6 weeks	Not given
	52	W	F	Mass in right axilla	5 weeks	3 years and well
	56	W	F	Diplopia; right supraclavicular node	Not given	Not given
Halsted	?	?	?	Mass in axilla	Not given	Not given
	?	?	?	Mass in axilla	Not given	Not given
	?	?	?	Mass in axilla	Not given	Not given
Jackson	62	W	F	Tumor in axilla	2 months	5 years and living
	37	W	F	Tumor in left axilla	1 day	2 years and well
	50	W	F	Tumor in left axilla	5 months	2 years and well
Kaplan and Reinstine	49	W	F	Mass in right axilla	3 months	2 years and died
	60	W	F	Mass in left axilla	4 weeks	4 years and died
	48	W	F	Mass in left axilla	Unknown	10 months and well
	53	W	F	Mass in right axilla, drawing sensation, shooting pain	3 weeks	1 year and died

TABLE I.—(*Continued*)

Reported by	Age	Race	Sex	Symptoms	Duration of Symptoms	Survival after Surgery
	35	W	F	Mass in left axilla, lymphadenitis	23 weeks	3 years and well
	42	W	F	Soreness & stiffness in left arm; painful axillary *scar*	3 weeks	1½ years and well
Primrose	43	W	F	Lump in axilla	Not given	Not given
Powell in Cogswell's paper	63	W	F	Mass in axilla	Not given	2 years and well
Rutkowski	40	W	F	Growth in axilla	6 months	1½ years & metastasis
	38	W	F	Growth under clavicle	3 weeks	Not given (died)
	43	W	F	Growth in right axilla	3 months	Not given (died)
	37	W	F	Growth in right axilla	Not given	Not given (died)
	38	W	F	Two growths in axilla	3 months & 2 months	Not given (died)
Shore	63	W	F	Several lumps in axilla	9 months	Not given
Stetten in Cogswell's paper	43	W	F	Mass in right axilla	Not given	13 years and well
	63	W	F	Lump in left axilla	Several yrs.	12 years
	59	W	F	Mass in left axilla	2 months	4 years and well
	46	W	F	Mass in right axilla	3 days	2 years
	44	W	F	Mass in right axilla	2 weeks	2 years
Westermeyer	58	?	F	Pain; small tumor in right axillary region	Not given	1 yr., good condition
	38	?	F	Small tumor in left axillary region	6 months	16 months, good condition
	52	?	F	Pain in left axilla and small tumor	Not given	5 years, good condition
	78	?	F	Small node in axilla	Not given	8 months, good condition
	48	?	F	Tightness & increase in volume in left axilla in '41; tumor in left breast in Feb. '42	Not given	4 years and died
	61	?	F	Small tumor in left axilla; small node in left breast	1 month	28 months, good condition

able breast mass. The other patient displayed a brawny edema of the lateral chest wall and the skin on the lateral aspect of the breast. This last case does not strictly fit the criteria for the term *occult carcinoma* and should not be included.

Since Halsted's first report, Cameron,[1] Shore,[11] Fitts and Horn,[3] Primrose,[9] Willis,[15] Cogswell,[2] and others[6, 7, 8, 10, 13, 14] have described this condition, which probably is more common than the literature would indicate, for many cases have not been reported. That this is true was brought out by Cogswell's report. In the discussion of the paper which he presented before the Western Surgical Association in 1948, several surgeons mentioned 1 or 2 cases which had come under their observation. To date, as far as we can determine, some 48 cases have been reported in the literature. We have summarized those which meet our criteria in table I.

INCIDENCE

Occult carcinoma of the breast is found more commonly in the older age group, most patients being in their fifth or sixth decade. No cases have been reported in males of any race. The reported cases have been predominantly of the white race.

CLINICAL ASPECTS

The clinical picture of an occult carcinoma of the breast is so variable and indefinite that a diagnosis cannot be made from symptoms alone. Actually, there is little that would suggest the possibility that the primary lesion is in the breast. One of our patients (case 4) complained of a pulling sensation in the axilla; of an itching in the region of the areola, and of not "feeling well generally." Fitts and Horn and Cogswell reported cases similar to ours in which the patients complained of a pulling and aching in the axilla of the affected side. Cogswell's patient also complained of a pain beginning in the axilla and radiating down the left arm and also to the breast. The most common clinical finding is an enlarged lymph node in either the axilla or supraclavicular area on the affected side. This was true in the majority of the reported cases and in all 6 in our series. Cameron described 1 patient who had swelling in the axilla with resultant skin changes such as redness and induration, followed later by softening and oozing, and still later, by the appearance of a mass in the breast which proved to be a carcinoma. Fitts and Horn reported a case in which the first finding of an occult carcinoma of the breast was orbital metastasis.

Willis, in his book *Pathology of Tumors*, cites a case in which hemorrhagic pericardial effusion was the first physical finding. Abdominal, visceral and peritoneal metastases gave the first indication of an occult carcinoma of the breast in 1 of Polson's patients. Thus, distant metastases to the regional lymph nodes, viscera or to bone may be the first finding in this condition.

PATHOLOGY

These lesions begin as very small, microscopic or macroscopic, deepseated adenocarcinomas and metastasize very early. The primary growth may remain so small as to be undetectable for many months. Sooner or later, if the patient

lives long enough, the mass surely will become evident in the affected breast. In the majority of the reported cases, after radical mastectomy had been done, a mass, usually of small *diameter*, was found in the resected specimen. However, this was not true in all cases. In case 3 of our series, after numerous sections, no gross lesion was found. It was only after a number of fixed sections had been made that several ducts in one section under the microscope were seen to contain clumps of anaplastic carcinoma cells. Fitts and Horn reported a case in which a node excised from the axilla showed the characteristics of a breast carcinoma, but even after a thorough search no primary growth was ever found. The importance of multiple serial sections of the resected specimen cannot be stressed too strongly when a gross lesion is not demonstrable.

Although in the reported series of cases the lesions were not always classified as to grade, the impression was gained that they represented a high level of intrinsic malignancy. In our series, none of the cases was below Grade III, the majority being Grade IV.

DIAGNOSIS

To establish an early diagnosis, all lymph node enlargements should be accounted for as early as possible. Excision biopsy is the quickest and most accurate means of establishing a diagnosis, and should be made without delay. Deep-seated pain in the region of the vertebral column or in other bones is often a symptom of metastasis from a breast carcinoma. A skeletal survey frequently will give a clue, especially if it shows a metastatic osteolytic lesion.

CASE REPORTS

Case 1. A white woman, 49 years of age, was admitted to Touro Infirmary on Feb. 21, 1944, with the chief complaint of a "lump in the right armpit," which had first been noted in December 1943. The patient did not complain of pain and noted that the mass had grown very little. There were no masses palpable in either breast and no discharge from the nipple. However, just below the border of the right pectoralis major muscle was a firm demarcated mass measuring 5 by 3 cm. No other nodes were felt.

On February 26, the mass was removed through an axillary incision. It was a large node just beneath the right pectoralis muscle. No other enlarged glands were found. The pathologist reported "anaplastic carcinoma, metastatic to lymph node." It was believed that the most likely origin was the breast. Since there were no masses in the breast, a radical mastectomy was not done, and the patient was treated with deep radiation therapy from March 6 to March 23, 1944.

She was followed at monthly intervals and was clinically well until April 1945, 14 months after the original procedure. At this time she developed some enlarged glands in the right axilla, just lateral to the scar. She was given four doses of deep radiation therapy to this area, which apparently controlled the recurrence. Even at this time no tumor could be found in the breast.

In July 1945, she developed enlargement of the left supraclavicular nodes and was given radiation therapy again. Another course on the left was repeated in about one month. This treatment gave some palliation. She gradually became more debilitated and died in 1946.

Case 2. This 60 year old white woman was admitted to Touro Infirmary on March 25, 1947, with the chief complaint of a "lump in the left armpit," which she had noted for the first time four weeks prior to admission. Eleven days after the appearance of the first mass, another appeared in the same area.

Physical examination revealed a very firm mass lateral to the left pectoralis major muscle. No masses were felt in either of the breasts. Hypertensive cardiovascular disease was an incidental finding.

On March 27, 1947, through an axillary incision, a mass measuring 4½ by 3 cm. was removed with several smaller nodes. The mass was adherent to the sheath of the axillary vessels but was successfully dissected off. A frozen section showed an adenocarcinoma arising from breast tissue. (The final report by the pathologist showed a Grade III metastatic adenocarcinoma, probably arising in breast tissue.)

Since no mass was present in the breast, it was decided not to do a radical mastectomy, but to give a course of deep radiation therapy. The radiation therapy, however, did not give much palliation, and the patient gradually went downhill. Her next admission to the hospital was on Dec. 12, 1950. At this time she had evidence of generalized carcinomatous metastases. There was a large ulcerated reddish purple mass in the left breast. Just below and behind the left ear there was *serous drainage*. The left breast at this time was described as being *almost solid* and there was brawny edema of the left arm. A left supraclavicular mass was also present. She showed anemia and weakness. She received at this admission further deep radiation therapy and such supportive measures as were considered necessary. However, she deteriorated and died on April 11, 1951.

Case 3. This 48 year old white woman was admitted to Touro Infirmary on July 29, 1953, with the chief complaint of postmenopausal bleeding of one week's duration. The patient had also developed a firm 2 by 2 cm. node in the left axilla.

On physical examination, the axillary node, measuring 2 by 2 cm., was palpated, as were some relatively enlarged nodes in the left posterior cervical region of the neck and one small node in the right axilla. Other physical findings were not contributory. On Aug. 2, 1952, the patient had a dilation and curettage and abdominal hysterectomy and salpingo-oophorectomy for her postmenopausal bleeding. At the same time, through an axillary incision, the node in the left axilla was excised. The report on this node was metastatic adenocarcinoma. No malignancy was found in the uterus, endometrium, tubes or ovaries. After the pathologist's report was received, a thorough search for the primary lesion was undertaken, with a high index of suspicion that the site probably was in the left breast. Bone marrow studies and gastrointestinal studies revealed nothing. Further palpation of the breasts disclosed no masses. When a biopsy of the left cervical and right axillary nodes proved negative, radical mastectomy on the left was done through a Stewart incision with a Rodman extension. The pathologist found, on serial sections, an extremely small area of ductal type carcinoma. Metastases to two other large axillary nodes were found.

The patient had a smooth postoperative course and has been in good health with no evidence of recurrence since her discharge 10 months ago.

Case 4. This 53 year old white woman was first seen on Jan. 25, 1951. At that time she said she had had shooting pains in the right breast for several weeks. The pain was described as radiating to the medial aspect of the area, and to the right neck. The past history revealed that in January 1947, a left radical mastectomy had been done for a Grade IV intraductal and infiltrating type carcinoma. She had no evidence of recurrence on that side at any time.

On physical examination a small firm mass in the right axilla just lateral to the border of the pectoralis major muscle was palpated. There were no other significant physical findings. The mass was not biopsied at that time. However, in May 1951, the patient went to the Mayo Clinic where she was operated upon on May 12. The mass, when excised, showed adenocarcinoma. A right radical mastectomy was done and a mass involving the pectoral muscles and fascia subjacent to the muscle was found. All axillary lymph nodes and lymph nodes within the pectoral muscle were involved. The final report from the pathologist was Grade IV adenocarcinoma. Postoperatively a course of deep radiation therapy was given. The patient got along relatively well until October of 1951, when a "BB"-sized, firm, nonfixed nodule appeared in the posterior portion of the right axilla associated with a thickening of the wall of the right axilla. The patient returned to the

Mayo Clinic where, on Oct. 18, 1951, this tumor was excised and found to be a Grade IV adenocarcinoma.

The patient did not do well following this procedure, and within the next month developed recurrent nodules over the right chest wall. These were excised on Nov. 15, 1951, at Touro Infirmary and reported as Grade III adenocarcinoma. The excised area was grafted with a split thickness graft. She showed progressive mental deterioration following this procedure and died two months later.

Case 5. This 35 year old white woman was admitted to Touro Infirmary on Feb. 16, 1951. At that time she complained of having had vague pain in the left axilla for several weeks. This pain had increased in severity, and five days prior to admission a mass was noted in the left axilla.

Physical examination showed a firm, tender mass in the left axilla, measuring 4 by 5 cm., which apparently was fixed to the deeper tissues. An associated interesting finding was the presence of dextrocardia. On February 20, the mass was removed through an axillary incision and the patient was discharged. However, the final sections showed a Grade III adenocarcinoma of a lymph node and a chronic suppurative lymphadenitis. The patient was readmitted on February 26, and a chest plate and gastrointestinal series were done. These showed no evidence of metastasis. On Feb. 28, 1951, a left radical mastectomy was done. Although the breast tissue was thoroughly examined, no tumor mass could be found. The axillary nodes in the specimen were not involved. The patient had a smooth postoperative course and was discharged. To the best of our knowledge she is living and well today.

Case 6: This 42 year old white woman was first seen on April 25, 1952 with the complaint of a painful linear area, apparently a scar, at the outer border of the left breast, with soreness and stiffness of the left arm. The patient could not account for the apparent scar; she had had no previous operation or injury to this area. She thought possibly the rubbing of her brassiere might have caused it. Examination of the breast and axilla was entirely negative except for the slight abnormality at the outer border of the left breast.

On April 28, tissue from the lesion was obtained under local anesthesia for biopsy. The pathologist's report was adenocarcinoma Grade III. On May 4, 1952, this patient was readmitted to the hospital. Again the physical findings were negative except for the recent scar due to the biopsy section incision. A left radical mastectomy was done. The pathologist reported scirrhous carcinoma, Grade IV, widely disseminated within the breast, and multiple metastases to the axillary lymph nodes. A small skin slough developed in the line of the incision. Irradiation was not given for this reason. After four months this area healed without a graft.

The patient did well until June 10, 1953, at which time she noticed a subcutaneous mass about 25 mm. in diameter at the point where the arm joins the axilla. This was biopsied and the pathologist reported metastatic carcinoma, Grade III. She was advised to have deep irradiation therapy and also irradiation to the ovaries. She went to Boston and there a wide excision of the site where the last biopsy section had been taken was done. Only fatty necrosis and scar tissue were found. Surgical sterilization was also done at this time. When last seen, Nov. 6, 1953, there was no evidence of recurrence.

TREATMENT

Three of our patients had radical mastectomies. One survived for 1 year and died; another, for 3 years and is well; and the last for 1 year and is well.

In the reported series, the majority of the patients had radical mastectomies. The average survival time was approximately 4.8 years. However, in this group several patients had especially long survival times, 2 living for 13 years, and another for 12, and yet another for 6.5 years.

The only conclusion we can draw from our cases and the reported series is that Haagenson's criteria[4] should be used to determine operability.

PROGNOSIS

The prognosis of this condition is not particularly good, although not entirely hopeless, and will depend upon the extent to which metastasis has taken place. Handley has shown that 46 per cent of the patients who have breast cancer with axillary nodal involvement will also have metastasis to the internal mammary chain. Wangensteen's figures[12] are even more discouraging. He says that 65 per cent of patients having axillary metastases will have either supraclavicular or internal mammary chain involvement. If the spread still is confined to the axillary nodes, the five year survival rate will be between 20 and 30 per cent.

Statistics tend to show that the five year survival rate can be increased by early radical mastectomy, or at least that generalized metastases and death can be significantly delayed in some of the patients who already have axillary spread. Apparently there is an appreciable length of time after axillary involvement has occurred before there is generalized spread of the disease. Therefore, early radical treatment will increase the life expectancy should the case meet the criteria of operability.

SUMMARY

Occult carcinoma of the breast has been defined.

The historic, clinical and pathologic aspects of this condition have been presented.

All reported cases have been classified in a table as to symptoms, duration, race, sex and survival time.

A series of 6 cases from Touro Infirmary has been reported.

Prognosis following radical mastectomy has been briefly discussed.

Haagenson's criteria of operability should be used.

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EDITORIAL

THE EFFECT OF ANTIBIOTICS ON SURGICAL INFECTIONS

The most serious defect in the clinical clerkship as a method of teaching is the fact that in any limited period several important clinical entities may fail to present themselves in the teaching wards and clinics. Therefore, most teachers periodically review the material that has been covered by their students and try to supply the deficiencies by some other method.

As the year 1952–1953 drew to a close, it dawned upon us that we had devoted entirely too little attention to several very grave surgical infections. Osteoperiostitis of the mandible and its deadly complication, Ludwig's angina, had not been mentioned. Our students were not aware of the gravity of infections of the dangerous area of the face—indeed they were not aware of the existence of such an area—and cavernous sinus thrombosis was to them only a name. Few of them had seen a case of empyema and all they knew about it was the rapidly fading memory of a lecture they had heard during the third year. Acute hematogenous osteomyelitis was to them a rather rare disease of no very serious import. Few if any of them realized that this disease, when not fatal, often leads to a life of semi-invalidism. Carbuncles in diabetics were, to many of them, comparable to a severe upper respiratory infection.

This observation led to a horrible suspicion. We are—and I think justifiably—proud of the clinical training that surgical residents get at the New Orleans Charity Hospital, but they see the same patients that the students see. After an impromptu sampling of the knowledge of our residents we found, to our gratification, that our suspicions were not fully justified, but that we still had no grounds for complacency.

During the past year I have had the opportunity of sampling the knowledge of young surgeons trained in various parts of the United States, and it is apparent that the deficiency is not limited to the *deep south*. One of my most vivid memories is that of Alfred Blalock's patient, persistent and futile efforts to find an American Board of Surgery Candidate who understood the surgical treatment of postpneumonic empyema. The reason for this difficulty was readily apparent. These young men had never seen a case!

It is obvious that this situation is due to the impact of sulfonomide and antibiotic therapy upon surgical infections. The young surgeons of today are, generally speaking, much better trained and better read than those of any previous generation, and they are at least as intelligent and industrious but they have seen few, if any, of the grave surgical infections mentioned above and with the aid of the antibiotics these few have been readily controlled.

We recently have taken 2-year samples from the records of the New Orleans Charity Hospital at 10-year intervals.

The incidence of acute empyema has dropped from 155 cases in 1931–32 to 23 in 1951–52 and the mortality rate from 18.7 per cent in 1931–32 and 39.3 per cent in 1941–42 to 0 per cent in 1951–52. The incidence of lung abscesses has actually increased but the mortality rate has dropped from 41.2 per cent to 17.8 per cent. Carbuncles of the face had a mortality rate of 10 per cent in 1931–

32 and 0 per cent in 1951–52. Erysipelas of the lower extremity showed a mortality rate of 28 per cent in 1931–32, 24 per cent in 1941–42, and 0 per cent in 1951–52. There were 35 cases of diabetic gangrene in 1931–32, 62 in 1941–42, and 158 in 1951–52. The mortality rates for these years were respectively: 37.1 per cent, 11.3 per cent and 5.06 per cent. Several of the infections discussed previously are not filed in the records under the names used, consequently they were not readily available for a hasty survey, but the above list will serve to illustrate the changing picture on our surgical service. Of course other factors have played important parts in the improvements noted, but one cannot doubt that the antibiotics and to a lesser degree, the sulfonomides, were the dominant factors.

This is a very happy state of affairs and would be grounds for undiluted optimism if it were not for the existence of strains of pathogenic micro-organisms that are insensitive to one or, on occasion, all of the antibiotics. Resistant strains always have existed and their number has been increased by the use of antibiotics. This is true even when these agents are used for legitimate reasons and in adequate dosage, but it is probable that they are developed much more frequently as a result of inadequate dosage employed in infections which do not justify their use. This indiscriminate use of antibiotics constitutes a very serious problem and should be combatted by every means at our disposal. Meleney reports that in his outpatient service the incidence of strains of organisms resistant to penicillin increased from 17.8 per cent in 1947–48 to 43.0 per cent in 1951. Curiously enough there was a drop to 31.1 per cent in 1952. It was also observed that the incidence of resistant strains is lower in outpatient services than in hospital wards, so there is evidence that the increase in the number of resistant strains is reaching alarming proportions.

Another serious difficulty that has become apparent is the fact that by the routine use of certain antibiotics, notably penicillin and streptomycin, we have frequently masked deep-seated infections such as perinephric and subdiaphragmatic abscesses. This has led to quite unjustified delay in instituting surgical measures.

Up to this time, new antibiotics have been introduced fast enough to keep pace with the incidence of strains resistant to the older ones, but one may be permitted to doubt that this can continue indefinitely. Furthermore, the culturing of the offending organism, and the testing of its sensitivity are not widely practiced.

As a result of these facts, if an individual is so unfortunate as to contract one of these very grave surgical infections caused by organisms that are insensitive to the antibiotics available, or at any rate to those employed in his case, and if he is treated by a surgeon unfamiliar with the behavior of such infections in the preantibiotic period, he is worse off than if these agents had never been discovered.

It seems obvious therefore that we *ancients* in the profession must continue to teach methods that the young men will consider outmoded. We must impress upon our students the fact that these deadly infections are not dead but sleeping, and that they still lie in wait for the unwary.

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